Environment Canada Environnement Canada

Guidelines for Surface Water Quality

Vol. 1 Inorganic Chemical Substances

SELENIUM

Adrian Demayo, Margaret C. Taylor and Stewart W. Reeder

INLAND WATERS DIRECTORATE,
WATER QUALITY BRANCH,
OTTAWA, CANADA, 1979.

(Résumé en français)

Contents

	Page
ABSTRACT	v
RÉSUMÉ	v
ACKNOWLEDGMENTS	vi
RECOMMENDED WATER QUALITY OBJECTIVES FOR SELENIUM	vii
SOURCES, OCCURRENCE AND FORM	1
Natural distribution of selenium	1
Production, consumption and uses	2
Forms of selenium	2
Pathways for entering the environment	3
RATIONALE	4
Raw public water supplies	4
Selenium in the human body	4
Essentiality of selenium in human health	5
Toxic effects of selenium in human health	7
Recommended objective for selenium in raw public water supplies	7
Combined effect of selenium and other elements and compounds on human and animal health	8
Aquatic life and wildlife	10
Movement of selenium through the aquatic ecosystem	10
Selenium levels in fish	10
Toxicity of selenium	10
Effect of selenium on the toxicity of other elements	11
Recommended objective for aquatic life and wildlife	12
Water supply for livestock	12
Essentiality of selenium to animal health	13
Selenium toxicity to animals	14
Recommended objective for water supply for livestock	15
Irrigation waters	15
Selenium in plants	15
Recommended objective for irrigation waters	16
Recreation and aesthetics	17
Recommended objective for recreational waters	17
Industrial water supplies	17
Recommended objective for industry	17
REFERENCES	17

Illustration

Figure 1. The cycling of selenium in nature	3

Abstract

A literature survey was carried out on the toxic effects of selenium and selenium compounds on human health, aquatic life, plants and livestock. The information is summarized in this publication. From it, maximum selenium concentrations in water at which toxic effects will not appear are recommended.

Résumé

On a effectué une étude de la documentation portant sur les effets toxiques du sélénium et de composés du sélénium sur les humains, les organismes aquatiques, les plantes et le bétail. Ces données sont résumées dans cette publication. En se basant sur ces données, on propose des valeurs des concentrations maximales de sélénium dans l'eau auxquelles on n'observe pas d'effets toxiques.

Acknowledgments

The Water Quality Objectives and Standards Group greatly appreciate the assistance of the many dedicated scientists located throughout federal agencies, especially Fisheries and Oceans Canada, Health and Welfare Canada, Agriculture Canada, Environment Canada, and National Research Council of Canada.

RECOMMENDED WATER QUALITY OBJECTIVES FOR SELENIUM

Uses	Recommended objectives (as total selenium)
Raw public water supplies	
Simple treatment for raw water	0.05 mg/L
Advanced treatment for raw water	0.25 mg/L
Aquatic life and wildlife	
Until further research has been done	0.01 mg/L
Agricultural water supplies	
Livestock watering	minimum 0.02 mg/L maximum 0.05 mg/L
Irrigation	Continuous use 0.02 mg/L Intermittent use 0.05 mg/L
Recreation and aesthetics	0.05 mg/L
Industrial water supplies	
Food and drink industries	0.05 mg/L where simple treatment for raw water occurs 0.25 mg/L where there is advanced treatment for raw water

Guidelines for Surface Water Quality
Vol. 1. Inorganic Chemical Substances. Selenium

Adrian Demayo, Margaret C. Taylor and Stewart W. Reeder

SOURCES, OCCURRENCE AND FORM

Natural Distribution of Selenium

The *Handbook of Chemistry and Physics* (CRC 1970, p. F-144) gives an abundance of 0.09 mg/kg of selenium in the earth's crust, which is the estimated average for igneous rocks. More recent data (Turekian and Wedepohl 1961; Taylor 1964) revised downwards the selenium concentration in igneous rocks to 0.05 mg/kg, based on a revised sulphur content of these rocks (down to 300 from 600 mg/kg) and the accepted sulphur to selenium ratio in igneous rocks of 6000. This places selenium as the 70th most abundant element after cadmium and silver but before other elements considered rare, such as palladium, platinum and gold. Actual analyses of igneous rocks showed a selenium concentration range of 0.004 to 1.5 mg/kg (Lakin 1973).

Some sandstones, limestones, shales (sedimentary rocks) and some meteoritic materials have been reported to contain selenium concentrations of over 100 to 200 mg/kg. Seleniferous soils are derived from shales. Shales are the parent material for approximately 40% of the earth's soils; sandstones, limestones, and igneous rocks, in equal amounts, are at the origin of the remaining 60%. Selenium concentrations in soils have been reported to range between 0.1 mg/kg, in a selenium-deficient area of New Zealand, and 1200 mg/kg, in an organic soil in Ireland (Johnson 1976). Phosphate rocks, and consequently some phosphate fertilizers, have been reported to contain 1 to 300 mg/kg selenium (NAS 1976, p. 10ff).

Some typical selenium minerals are clausthalite, PbSe; crookesite, $(Cu,Tl,Ag)_2Se$; eucairite, $(Cu,Ag)_2Se$; and naumannite, (Ag,Pb)Se. The metal selenides are nearly all isomorphous with the corresponding sulphides, which explains their occurrence with copper, lead and iron sulphides (Bagnall 1973).

There are a number of estimates of selenium concentrations in seawater, which range from approximately 0.00009 mg/L (Bowen 1966, p. 20) to approximately 0.006 mg/L (Sillén 1963). Actual data obtained by Schutz and Turekian (1965) showed concentrations of 0.000052 mg/L in the Antarctic Ocean, 0.00010 to 0.00012 mg/L in the Caribbean Sea, and 0.000076 to 0.00013 mg/L in the North Atlantic Ocean. It is interesting to compare these with Greenland glacial ice concentrations of 0.008 to 0.0142 mg/kg (Lakin 1973).

The available selenium data for fresh waters is more consistent than for seawater, showing little difference between the average selenium content of rivers draining non-industrialized areas and rivers draining, for example, mining or coal regions. The Amazon River contains 0.00021 mg/L; the Rhone River at Avignon (France), 0.00015 mg/L; and the Susquehanna River, Pennsylvania, in a coal-mining industrial region, 0.000325 mg/L (Lakin 1973). Some rivers in Germany have concentrations ranging from 0.00012 to 0.0015 mg/L (Traversy *et al.* 1975). Selenium concentrations in surface waters seem to be pH dependent. Data from Colorado surface waters showed that at pH 6.1 to 6.9, selenium concentrations were below 0.001 mg/L. At pH values of 7.8 to 8.2, selenium concentrations ranged from 0.001 to 0.4 mg/L. At pH 6.3 to 6.7, selenium is precipitated quantitatively as ferric selenite $[Fe_2(SeO_3)_3]$. At a pH of about 8.0, selenite may be oxidized to the soluble selenate ion SeO_4^{2-} (Lakin 1973).

In the Great Lakes region the selenium concentrations varied between <0.0001 and 0.0008 mg/L for lake waters and between <0.0001 and 0.0006 mg/L for river waters. In both cases the average concentration was <0.0001 mg/L, which was the detection limit of the analytical method used to analyze the samples. The sediments had much higher concentrations: 0.20 to 2.00 mg/kg (Traversy *et al.* 1975).

The concentration of selenium in precipitation seems to be highly variable. Precipitation in the Great Lakes region showed concentrations from <0.0001 to 0.001 mg/L (Traversy *et al.* 1975). Precipitation samples (rain and snow) collected over a period of 18 months in the Perch Lake basin, a remote area of Ontario, showed selenium concentrations between 0.000015 and 0.00065 mg/L (Merritt 1976). Rainwater samples from eight locations across the United States gave results ranging from 0.00002 to 0.00016

mg/L in the water, and 0.00004 to 0.0002 mg/kg in the particulate matter (Kubota *et al.* 1975). Snowfall concentrations in Cambridge, Mass., were from 0.09 to 0.27 mg/L (Lakin 1973).

Rainwater from six locations in Denmark had selenium concentrations ranging from 0.00011 to 0.00039 mg/L. In general, the higher concentrations were observed in the lower rainfalls (Kubota *et al.* 1975). Precipitation samples in the United Kingdom had concentrations of 0.0002 to 0.0016 mg/L (Merritt 1976).

It is interesting to note that the chemical analysis of air particles collected over the North Atlantic at 8 to 20 m above sea level, and at the geographic South Pole, at 2300 m above sea level, showed very high enrichment factors for selenium. Enrichment factors of 10 000 to 18 000 over the concentration expected from the crustal composition and crustal weathering processes around these locations were found. It has been suggested that the anomalously high atmospheric concentration of selenium originates primarily in natural high temperature sources, such as volcanism, and in biological mobilization (Zoller *et al.* 1974; Duce *et al.* 1975).

Production, Consumption and Uses

Selenium is produced commercially from the anode slime deposited during the electrolitic refining of copper by pyrometallurgical methods and from flue dusts from copper and lead smelters. Thus the quantity produced is a function of the level of production in the world primary copper refining industry. The non-communist world production of selenium in 1974 was 1097 tonnes, of which Japan produced 363 tonnes, Canada 268 tonnes, United States 229 tonnes, etc. In Canada the total production of refined selenium in 1974, including imported material and secondary sources, amounted to 334 tonnes, of which 268 tonnes were from Canadian copper refineries and domestic primary materials. By province, Quebec produced 184 tonnes and Ontario, 56 tonnes.

The consumption of selenium in Canada in 1974 was approximately 14 tonnes. During that year 421 tonnes were exported, mainly to the United States and United Kingdom (93%) (Wood 1975).

Selenium is used in the manufacture of glass, steel, electronic components, explosives, animal and poultry feeds, fungicides, pigments and xerography. In Canada its main use (73%) is in glass manufacturing. Small quantities of selenium neutralize the greenish tinge caused by the iron impurities in sand. When larger quantities of selenium are added to the glass batch, the brilliant ruby-red glass used in traffic lights, decorative tableware, etc., is produced. Selenium is also used in the production of tinted glass used in many new buildings to lower heat conductivity. Added to stainless steel (0.2 to 0.35%) selenium improves machinability, and iron selenide added in small quantities (0.01 to 0.05%) to steel prevents pinhole porosity during casting. In the pharmaceutical industry, selenium is used in the manufacture of nicotinic acid, cortisone and certain drugs for the control of dermatitis and other diseases in humans and animals (Wood 1975).

Forms of Selenium

The possible oxidation states for selenium are -2, 0, +4 and +6. The +2 oxidation state has not been found in nature. Selenium in the +6 oxidation state (selenate) is stable in alkaline, oxidizing conditions. Soluble selenates therefore would be expected in alkaline soils, and probably it is in this form that selenium uptake by plants takes place. Thus, selenate appears to be an important form of selenium with regard to environmental pollution. Of the compounds in which selenium is in the +4 oxidation state (selenite), ferric selenite is important in the environmental cycle of selenium, as it has a very low solubility and adsorbs on soil particles, consequently lowering the availability of selenium to biota. Under reducing and acidic conditions, the formation of very insoluble elemental selenium is favoured, thus decreasing again the availability of selenium which enters the environment in the selenate or selenite form. Mild reducing agents, such as SO_2 or ascorbic acid, convert selenite to selenium. It appears that elemental selenium is a major "inert" sink for selenium entering the environment in various ways. Further reduction of selenium leads to selenide (Se^{2-}). Hydrogen selenide (H_2Se) fumes are toxic, but in air the compound decomposes rapidly to give elemental selenium and water, thus there might only be a danger of exposure to H_2Se in some industrial installations. Selenides of heavy metals, mercury in particular, are very insoluble. The formation of mercuric selenide may be a major detoxification mechanism of methylmercury by dietary selenite (NAS 1976, p. 1ff).

The forms of selenium which occur in living systems depend on the form and the amount of selenium supplied to the organism. If these compounds are classified by their molecular weight (Ganther 1974), then in the low molecular weight group, compounds such as Se-methylselenocysteine, Se-methylselenomethionine, dimethyl selenide, selenocystine, selenomethionine, etc. are included. They occur in free form and have been identified and isolated from various plants and animals. Brinckman and Iverson (1975) quoted studies showing the formation of dimethyl selenide from soils rich in selenium by several fungal cultures. Chau *et al.* (1976) reported the formation of dimethyl selenide and

dimethyl diselenide in both soil and sediment samples enriched with a variety of selenium compounds: sodium selenate and selenite, selenocystine, selenourea, and seleno-DL-methionine. The production of the two selenides was associated with microbiological growth.

A large part of the selenium occurring in plant and animal tissues is associated with protein. The selenium replaces the sulphur in sulphuraminoacid and forms a covalent bond with carbon, or is attached to sulphur atoms of cysteine residues. There is evidence that the inorganic selenium from selenates and especially selenites is incorporated in selenoamino acids and proteins by plants and animals (Ganther 1974). Animal cells in the presence of vitamin E have the ability to convert selenium from +6 to -2 valence state and apparently use Se^{2-} as a catalyst. Glutathione peroxidase, an Se^{2-}-dependent enzyme, is involved in the union of hydrogen and oxygen, the final step in the respiratory chain (Frost and Lish 1975).

Pathways for Entering the Environment

A schematic representation of the selenium macrocycle in the environment is given in Figure 1. Quantitative estimates for the amount of selenium associated with each path represented in Figure 1 are almost non-existent.

It has been estimated that 7200 tonnes of selenium enter the environment by weathering (Bertine and Goldberg 1971). Kharkar *et al.* (1968) have shown that part of the stream supply of selenium to the oceans is made up of dissolved selenium and selenium adsorbed on the suspended materials. This latter part is probably desorbed once it

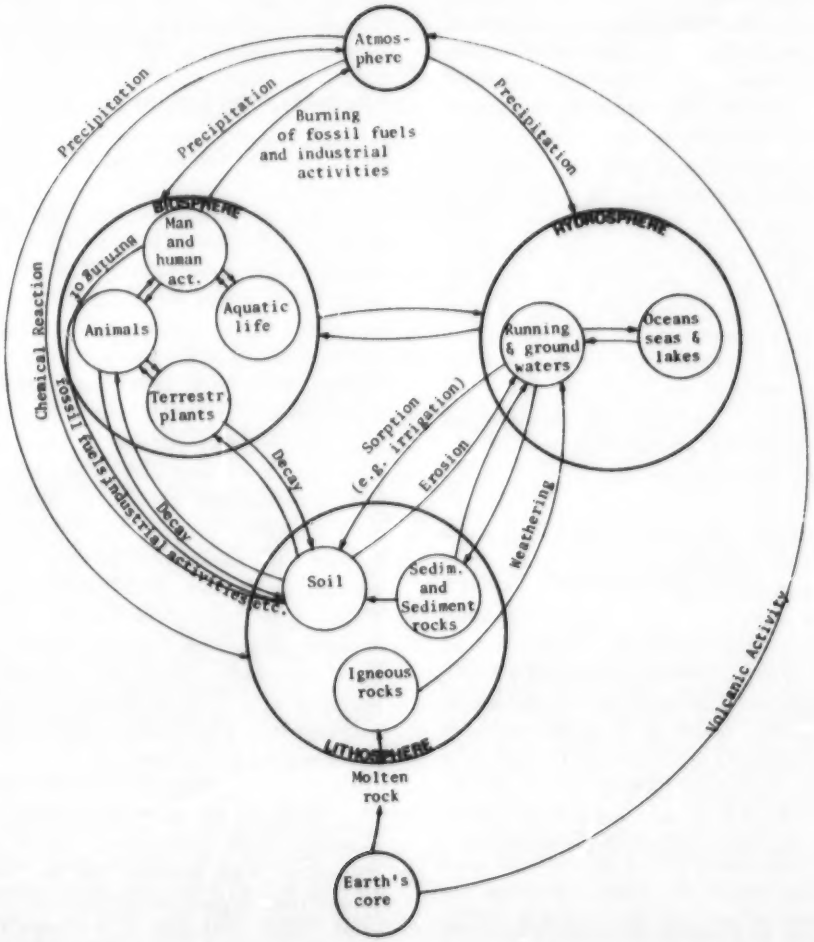

Figure 1. The cycling of selenium in nature (adapted from NAS 1976, p. 72).

reaches the sea through displacement of the selenium ions by magnesium and sodium ions present in sea water in high concentrations. The ratio of the two parts varies with the nature of the suspended sediments, the absolute concentrations of selenium in river water and on suspended sediments, and the flow. It has been estimated that the dissolved selenium in river water contributes about 80% and the suspended sediment 20% to the amount of dissolved selenium in seawater (Kharkar *et al.* 1968). Estimates are lacking for the amount of selenium entering the environment by volcanic activity, another major natural process.

Among the human activities, the major contributor to selenium entering the environment is the combustion of fossil fuels, i.e. coal and oil. The average selenium concentration in coal ranges from approximately 0.1 to around 5 mg/kg (Demayo *et al.* 1979). The world consumption of coal in 1974 (lignite, bituminous, and anthracitic) was approximately 3 billion tonnes, of which about 50% was used for producing energy (McMullen 1976; Bertine and Goldberg 1971). In the United States the amount of selenium released from coal used for power production has been estimated at 14% of the total selenium content of coal (Klein *et al.* 1975). This estimate took into consideration the existence of pollution control equipment and the fact that selenium is enriched in the fly ash which is released to the atmosphere compared to the bottom ash or to the ash which is retained in the air pollution control equipment, e.g. precipitators. Globally the percentage of selenium being emitted to the atmosphere is probably greater than the value for the United States. Using a value of 25% and an average concentration of selenium in coal of 2.5 mg/kg, it can be calculated that annually approximately 950 tonnes of selenium are released to the atmosphere from the burning of coal and that another 2800 tonnes of selenium enter the environment through the disposal of ash resulting from coal burning.

The selenium content of crude or fuel oils is much lower than that of coal. Analysis of 47 oil samples from various parts of the world gave results ranging from <0.006 to 2.2 mg/L with an average of approximately 0.6 mg/L (NAS 1976, p. 23). Averages estimated or obtained by other workers were 0.17 mg/L (Bertine and Goldberg 1971), 0.92 mg/L for crude oils and 0.99 mg/L for heavy petroleum (Hashimoto *et al.* 1970). Assuming a concentration of 0.6 mg/kg, the estimated world production in 1976 of crude oil of 2.47 billion tonnes contained 1300 tonnes of selenium. Presumably all this selenium is released into the atmosphere by the burning of oil and to other parts of the environment by oil spills.

The contribution to selenium emissions from other sources has been estimated to be small. Thus, for the year 1970, mining, smelting, and refining industries plus other industrial activities (glass, electronics, etc.) released an estimated 360 tonnes (NAS 1976, p. 46). Thus, the total annual selenium emissions from human activities are around 5400 tonnes.

World production of selenium in 1969 was 1480 tonnes and in 1970, 1300 tonnes (NAS 1976, p. 32). Therefore, the selenium emissions from human activities are greater than the world production of selenium by a factor of approximately three.

Microcosm experiments have shown that after 56 days about 75% of the selenium applied to the terrestrial part of the microcosm via simulated rainfall was retained in the soil; terrestrial plants retained about 8%, and litter, 6%. The rest reached the aquatic environment where sediments retained 8 to 9% and the combined water, fish, snails, periphyton, etc., 0.8% (Huckabee and Blaylock 1974).

RATIONALE

Raw Public Water Supplies

Selenium in the Human Body

The so-called "reference man" contains 13 mg of selenium (Schroeder 1974, p. 22). The concentration in various organs ranges from 0.05 to 0.92 mg/kg of the whole tissue in an infant and from 0.10 to 1.24 mg/kg in an adult. The highest concentrations in both adult and infant are encountered in the kidney and thyroid gland. Although the liver and muscle have lower concentrations (0.34 and 0.31 mg/kg in an infant, and 0.39 and 0.40 mg/kg in an adult, respectively), because of the weight of these tissues they store the largest part of the selenium in the human body (Dickson and Tomlinson 1967). Apparently there is little difference between the blood level of selenium of humans from seleniferous (average 0.256 mg/L) and non-seleniferous (average 0.157 mg/L) areas (Allaway *et al.* 1968). Recent data show that the selenium concentration in blood can reach much higher values without any reported toxic symptoms. Thus, for example, data quoted by Schrauzer *et al.* (1977a) showed that human blood samples collected from a seleniferous area in Venezuela were found to contain 0.7 to 1.5 mg/L.

Selenium can enter the human body via the gastrointestinal tract, respiratory system and through skin absorption. Rats absorbed 95 to 100% of the selenium administered in diets containing 0.02 to 4.02 mg/kg (Brown *et al.* 1972). Although it is known that certain volatile selenium compounds are toxic, e.g. H_2Se, no data were

found in the literature about the absorption of selenium via the respiratory tract. Regarding the absorption through skin, experiments have shown that 10% of the selenium of a 0.1 mol/L sodium selenite (7900 mg Se/L) solution applied to rat skin was absorbed in one hour (Dutkiewicz *et al*. 1972).

The absorbability and the activity of selenium compounds are a function of their chemical form. The most active selenium compound is probably Factor 3, which is found in crude casein, kidney and liver powder or brewer's yeast and in which organically bound selenium is the essential component (Schwarz and Pathak 1975). In foods selenium is always organically bound. However, not all organically bound selenium is as active as that from Factor 3. Experiments with rats suggested a higher absorption of selenium from selenites and selenates than from selenides, the elemental form, or some organic selenium compounds, e.g. selenodiacetic acid or selenopropionic acid (Underwood 1971, p. 330). Factor 3 prepared from pork kidney powder was 50% effective against dietary liver necrosis at dosages (ED_{50}) supplying 0.007 mg of selenium per kilogram of diet. Selenium from selenites had a 50% effective dose (ED_{50}) of 0.022 mg/kg of diet, while ED_{50} for phenylselenide was 1.34 mg/kg of diet and selenium in elemental form (grey modification) was considered inactive (Schwarz and Pathak 1975).

Absorbed selenium is at first carried mainly in the plasma, apparently in association with plasma proteins, from which it enters all tissues (Underwood 1971, p. 331).

Excretion of selenium takes place via urine, feces, expired air, sweat and milk. The amounts and proportions depend upon the level and the form of the intake, the nature of the rest of the diet, and the presence of other elements, notably arsenic. Most of the selenium in the feces consists of the selenium which has not been absorbed from the diet, together with small amounts excreted into the bowel with the biliary, pancreatic and intestinal secretions. Urine is a major elimination path, and at high dietary intake exhalation of selenium becomes more and more important (Underwood 1971, p. 332). The normal selenium concentration in urine should be below 0.100 mg/L. Values of up to 1.98 mg/L have been reported in seleniferous areas or in cases of industrial exposure (Cooper and Glover 1974).

Essentiality of Selenium in Human Health

Until about twenty years ago selenium was identified as a toxic element to plant, animal and human life. Since then, a selenium deficiency has been recognized in plant and animal life (Allaway *et al*. 1974). In 1957 selenium was detected as an integral constituent of Factor 3, an agent prepared from pork kidney powder that prevents liver necrosis. Dietary liver necrosis in rats is produced by simultaneous absence of selenium and vitamin E from the diet (Schwarz and Pathak 1975).

The role played by selenium in human health, however, is still not well understood, but developments are occurring rapidly in this field. Apparently selenium plays a role in the protection of cell and cell membranes from oxidative damage; facilitates the union between oxygen and hydrogen at the end of a metabolic chain and the transfer of ions across cell membranes; and participates in the synthesis of proteins, e.g. immunoglobulin and ubiquinone. Selenium deficiency, for example, has been correlated with decreases in glutathione peroxidase. This is an important enzyme in destroying H_2O_2 and organic hydroperoxides such as lipid hydroperoxides; it also guards against oxidative damage to cell membranes and other oxidant-sensitive sites in the cell (Hoekstra 1975; Frost and Lish 1975). It has been suggested that selenium might aid both hydrogen removal and oxygen addition in the metabolism of foodstuffs. The rapid and reversible *in vivo* conversion of selenoxides (Se^{4+}) to selenides (Se^{2-}) suggests a role for selenium in maintaining the oxidation-reduction potential and the pH equilibria within cells (Frost and Lish 1975).

Data quoted by the United States National Academy of Sciences (NAS 1976, p. 92) show that the average diet in the United States contains 0.06 mg of selenium per day or, using the weight of the reference man of 70 kg, 0.025 mg per kilogram of body weight per month. For comparison, a selenium-adequate rat diet contains 0.06 mg per kilogram of body weight per month. In Canada the daily intake from an average diet has been estimated at 0.068 mg (Neri 1976). Other workers estimate the selenium intake at 0.134 mg/day in the United States and 0.151 mg/day in Canada (Watkinson 1974) or at 0.167 and 0.169 mg/day, respectively (Schrauzer *et al*. 1977a).

The whole body half-life for selenium has been estimated at 11 days (Buhler 1972).

If one accepts the results of animal studies as valid indicators of the nutritional needs of man, it has been estimated that the human dietary requirement is 0.1 to 0.2 mg of selenium in a daily diet of 1 kilogram (Oldfield *et al*. 1971). Others (Schrauzer *et al*. 1977a) estimated the daily selenium requirement at 0.2 to 0.3 mg, and a maximum intake of 0.5 mg/day has been suggested for the maintenance of good health (Sakurai and Tsuchiya 1975). Data (NAS 1976, p. 20) show that in the American diet, the seafoods contain the highest selenium concentration (0.337–0.658 mg/kg) followed by other high-protein foods: meat (excluding kidney), 0.116 to 0.432 mg/kg; egg yolk, 0.183 mg/kg; etc. Cereals can contain substantial amounts of

selenium (range, 0.026–0.665 mg/kg), but they are not reliable sources, since their selenium content depends on the level of available selenium in the soils where parent grains were grown. Vegetables and fruit (<0.002–0.039 mg/kg), with the exception of garlic (0.249 mg/kg) and mushrooms (0.118 mg/kg); milk (0.012 mg/L) and dairy products are poor sources of selenium. Beer, with a concentration of 0.19 mg/L, is a good source of selenium (Schroeder 1974, p. 123). Ordinary cooking practices may cause some selenium losses, but for most food the losses are minor (Higgs *et al.* 1972). Refining of the whole product (wheat flour, sugar, milk, etc.), however, leads to very high or complete loss of selenium (and other nutrients) (Schroeder 1974, p. 121).

Direct evidence for the role of selenium in human nutrition is lacking, but a number of diseases have been connected with low selenium intake. Children suffering from kwashiorkor (disease caused by insufficient dietary protein affecting chiefly children in tropical areas; the symptoms are diarrhea, edema, partial loss of pigmentation, desquamation and stunted growth, in severe cases leading to death) had selenium blood levels of 0.1 mg/L compared with 0.23 mg/L in well-nourished children. Selenium supplementation improved the health of the children and the selenium blood levels increased to control levels (NAS 1976, p. 93). *In vitro* experiments showed an enhanced uptake of radioselenite by red blood cells from children with kwashiorkor (Burk *et al.* 1967).

Recently, selenium has been implicated in diseases of the circulatory system. In a study in the United States, the age-specific death rates for cardiovascular, renal, cerebrovascular, coronary, and hypertensive heart disease were all significantly lower in a very high selenium area than in a low selenium area. Rats and lambs fed selenium-deficient diets developed abnormal electrocardiograms (Shamberger *et al.* 1975). Also, inverse relationships between selenium blood concentration and age-specific (55 to 64) death rates from cardiovascular-renal, coronary and hypertensive heart diseases were found in another study conducted in the United States (Shamberger *et al.* 1979). In the same paper it is mentioned that in China keshan (congestive heart) disease was eliminated by administration of 1 mg of sodium selenite per week for a period of 2 years. Keshan disease affects children in areas severely deficient in selenium. Similarly, selenium deficiency, which is prevalent in Finland, is thought to underline the high cardiac and cancer mortality rates in that country (Marjanen and Soini 1972). Capsules containing selenium (as selenite) and tocopherol (vitamin E) are used for the relief of some aspects of heart disease, e.g. anginal pain (Frost and Lish 1975).

Although not conclusive, epidemiological studies seem to indicate an inverse relationship between selenium exposure and mortality from gastrointestinal cancer in the United States. This was consistent with the protection against chromosomal breakage by antioxidants such as sodium selenite (Na_2SeO_3), butylated hydroxytoluene (BHT), ascorbic acid (vitamin C), dl-α-tocopherol (vitamin E) in *in vitro* experiments. It has been proposed that selenium in the cancer process prevents the peroxidative attachment of the carcinogen to DNA. It has been estimated that the selenium intake must be a higher level, 1 mg/kg of diet, to counteract the carcinogenic effects of certain compounds compared with the level necessary to prevent selenium deficiency which was necessary at 0.1 mg/kg of diet (Shamberger 1974; Shamberger *et al.* 1973).

A cancer mortality study using data from 27 countries has shown significant inverse correlations between dietary selenium intake and the age-corrected mortalities from cancer of the colon, rectum, prostate, the female breast, ovary, lung (male), and from leukemia. It has been suggested that if dietary selenium intake were increased to 0.2 to 0.3 mg/day, i.e. about twice the current U.S. value, cancer mortalities should decline significantly (Schrauzer *et al.* 1977a). It has also been suggested that the lower incidence of breast cancer in populations of Japan, India and Singapore compared to the United States is due, at least in part, to the high selenium and low polyunsaturated fatty acids (PUFA) content of non-western diets. Selenium has been shown to prevent peroxidative damage from PUFA in tissues through its role as a component of the glutathione peroxidase enzyme, which is present in human breast tissue (Wilson and Petrakis 1976).

Studies by Money (1970-71) suggested that the sudden-infant-death syndrome might be due to combined selenium and vitamin E deficiency in cow's milk, or that, at least, the deficiency played a secondary role (Rhead *et al.* 1972). Breast-fed infants received ten times more vitamin E and two times more selenium than babies fed on cow's milk.

Selenium deficiency has also been connected with the finding of cataracts in rats, and selenium appeared to be needed for the motility of sperm (NAS 1976, p. 89ff). Selenium and vitamin E stimulated antibody formation in rabbits and enhanced the immune response in mice (Spallholz *et al.* 1973, 1974). Selenium-tocopherol capsules are used in veterinary medicine for treating clinical signs of arthritis. This provides a useful background for evaluation of the selenium role in human arthritis.

Toxic Effects of Selenium in Human Health

The acute toxicity of selenium compounds affects primarily the central nervous system. The symptoms produced by injection of most selenium compounds is similar to that of strychnine poisoning (Schwarz and Pathak 1975). One of the first signs of chronic poisoning by selenium in humans is brittleness, malformation, discolouration, and loss of fingernails. Selenium has an affinity for nails because of their high content of sulphur-containing amino acids cystine and methionine. Other symptoms include chronic dermatitis and loss of hair. Inhalation of selenium dust, hydrogen selenide, or selenium dioxide powder, which might occur in industrial situations, is characterized by severe irritation of the respiratory system. Signs such as painful nail beds, metallic taste, garlic odour of the breath, nervousness, mental depression, and anemia might indicate chronic selenium poisoning (Weswig 1973; NAS 1976, p. 116). Only one case of acute poisoning of humans by selenium has been reported so far (NAS 1976, p. 118). Several cases of chronic poisoning have been reported. One case of chronic poisoning involved the consumption of well water containing 9 mg Se/L. Five children of 6 months to 10 years old were affected. It was the first case reported of selenium intoxication from ingestion of drinking water (Beath 1962).

Apparently selenium is also capable of increasing dental caries when consumed during the period of tooth development (Hadjimarkos 1973). Children in high selenium towns had a significantly higher prevalence of caries than children in low selenium towns. Milk was used as an indicator of selenium availability in the area. The mean selenium content of milk samples produced and consumed was 0.045 mg/L in high selenium towns and 0.016 mg/L in low selenium towns (Ludwig and Bibby 1969). It has been proposed that incorporation of dietary selenium into the protein fraction of the enamel during the development of teeth inhibits mineralization of this tissue, resulting in increased susceptibility to caries (Hadjimarkos 1973).

Experimental studies using rats have shown that the body weight decreases at daily doses of 0.5 mg of selenium as selenite or selenate, and that liver lesions are caused by concentrations of 0.25 mg/kg of diet. The minimum toxic level for lesions of kidney, heart and spleen has been estimated at 0.75 mg/kg of diet (NAS 1976, p. 114; Harr and Muth 1972). Harr and Muth (1972) have also concluded that the dietary threshold for physiologic-pathological effects is 0.4 mg/kg and for pathologic-clinical effects, 3 mg/kg.

Selenium is not cumulative in the tissues. At very high selenium intake, 10 to 100 times the normal intake, tissue selenium concentrations rise until they reach levels of 5 to 7 mg/kg in the liver and 1 to 2 mg/kg in the muscles, i.e. 5 to 10 times the normal levels in these organs. Beyond these levels excretion begins to keep pace with absorption (Underwood 1971, p. 325).

The toxicity of selenium has been attributed to an over-accumulation of selenite ions, which act as oxidants to inhibit SH interactions. Such toxicity is readily avoided or reversed (Frost and Lish 1975).

Most selenium compounds are toxic even when they are effective in the prevention of dietary liver necrosis. Besides the amount and intake route, it was found that the most important difference between properties that determine toxicity and essentiality is in the area of solubility. The lipid-water distribution coefficient played a critical role in acute toxicity, whereas it was irrelevant in the prevention of liver necrosis. The acute toxicity of selenium compounds is directly related to their lipid solubility. Again for rats, the "therapeutic index" (ratio of LD_{50}/ED_{50}) ranged between 70 for di-seleno-4, 4^1 -di-(2, 2-dimethyl) butyric acid and 20 500 for monoseleno-di-acetyl asparagine (Schwarz and Pathak 1975).

Recommended Objective for Selenium in Raw Public Water Supplies

The published information shows that selenium is probably both an essential and toxic element. Its essentiality and toxicity are functions of the nature of the species affected, the nature of the selenium compound, the intake route, the amount of selenium ingested, and the composition of the diet. The standards for domestic water supplies and the drinking water standards in the United States, Australia, Canada, USSR, and the World Health Organization recommend a maximum limit of 0.01 mg/L for selenium (Rail and Hadley 1976; Hart 1974, p. 51; NHW 1968; Eriksson 1977). In Sweden for waters purified according to "normal" practice (chemical flocculation and slow filtering) the limit is 0.01 mg/L, and for waters which are much more extensively treated before use, the limit is 0.05 mg/L (Eriksson 1977).

Simple water treatment processes remove small amounts of selenium. Thus, in a pilot plant, sand filtration removed 9.5% and lime coagulation-settling process, 16.2% of approximately 0.013 mg Se/L (as SeO_3^{2-}) or less. Advanced treatment consisting of sand filtration, activated carbon absorption and cation exchange removed 44.7% of selenium from the same influent. Addition of an anion exchange step raised the removal to 99.9% (Linstedt *et al.* 1971). Similar results, 67 to 95% removal with advanced treatment, were reported by Cohen (1977). Thus, lime

precipitation plus activated carbon treatment removed 67% of an initial concentration of 0.06 mg/L (effluent concentration 0.02 mg/L) and 95% of an initial concentration of 0.5 mg/L (effluent concentration 0.025 mg/L). The effluent from a ferric chloride-activated carbon treatment had a concentration of 0.013 mg/L (initial concentration 0.05 mg/L) (Cohen 1977). Secondary municipal sewage treatment plant effluents containing 0.002 to 0.009 mg/L have been reported. When this effluent was subjected to a tertiary treatment consisting of lime treatment of pH 11 in a rapid-mix basin, flocculation, sedimentation, ammonia stripping, recarbonation, mixed-media filtration, activated carbon absorption, and chlorination, the selenium concentration was reduced from 0.009 mg/L to 0.001 mg/L. When the selenium concentration of the influent was lower, 0.002 to 0.003 mg/L, the additional treatment had little or no effect (Argo and Culp 1972).

Pletnikova (1970) reported that selenium in concentrations of 0.01 to 0.025 mg/L imparted a marked earthy odour to drinking water, probably owing to the formation of volatile esters and dialkyl selenides. In concentrations of 0.1 mg/L, selenium imparted an astringent taste to water.

Studies suggest that human daily selenium intake should be at least 0.2 to 0.3 mg, with some suggestions of an intake as high as 3.0 mg (Schrauzer *et al.* 1977a; Allaway 1975) to prevent selenium deficiency and the related health problems, e.g. cancer, hypertension. The present Canadian intake has been estimated at 0.068 to 0.151 mg/day (Neri 1976; Watkinson 1974). If 0.2 mg/day is accepted as the minimum daily requirement of selenium and if, arbitrarily, 20% of this is allowed to come from the drinking water, then the minimum selenium concentration of the drinking water should be 0.02 mg/L, assuming a consumption of 2 L of water per day.

In cases where raw waters undergo only a simple treatment, a maximum total selenium concentration of 0.05 mg/L is recommended. For those waters undergoing advanced treatment, a maximum total selenium concentration of 0.25 mg/L is acceptable.

Combined Effect of Selenium and Other Elements and Compounds on Human and Animal Health

Data from 28 countries have shown that the per capita intake of zinc, cadmium, copper and chromium correlated with the age-corrected mortalities from leukemia and cancer of the intestine, prostate, breast, skin and other organs. In particular, mortalities from cancer of the intestine, rectum, breast, ovary and lungs correlated directly with zinc blood concentrations and inversely with selenium blood concentrations. An inverse correlation was also found between zinc and selenium blood levels suggesting an antagonism between these two elements. Zinc appears to counteract the cancer-protecting effect of selenium in humans, possibly by lowering the physiological activity of selenium or by preventing selenium uptake. In large doses zinc produces symptoms of selenium deficiency (Schrauzer *et al.* 1977b).

The same antagonistic effect exists for the cadmium-selenium pair. Selenium reduces cadmium toxicity, e.g. testicular necrosis or other effects related to reproduction, and prevents the teratogenic effects of cadmium. Administration of cadmium salts decreases the respiratory excretion of volatile selenium compounds (Parizek *et al.* 1974). Animal experiments have also shown that cadmium-induced hypertension can be reversed by the addition of the half-molar equivalent of selenium (Perry *et al.* 1974). It has been suggested (Parizek 1976) that a reaction between the thiol groups of proteins and selenium and cadmium is responsible, or that the decreased toxicity of cadmium is due to the readily formed insoluble cadmium selenide (CdSe) (Schrauzer *et al.* 1977b).

No synergistic effect was detected when a selenium-cadmium mixture was injected into the chick egg's yolk. The reduction in the hatchability of the eggs agreed very well with that calculated from the hatchability frequencies of individual metals (Birge *et al.* 1976).

In experiments with acute intoxication by inorganic mercury salts, the typical pathological changes did not appear in animals given selenium compounds. Selenium compounds, in protecting experimental animals, increased the retention of mercury in the organism and changed its distribution within the body (Parizek *et al.* 1974). For example, selenium decreased the proportion of ingested mercury (as methylmercury chloride) deposited in livers of chicks by nearly 50% compared with chicks fed mercury alone. In contrast, more mercury was found in brains of chicks fed mercury and selenium than in brains of birds fed mercury alone (Sell and Horani 1976).

The administration of selenium compounds decreased the passage of mercury into fetuses and milk but increased the level of mercury and cadmium in the blood and blood plasma by several orders of magnitude. The chronic toxicity of methylmercury is also modified by selenium. The addition of 0.5 mg Se/kg (as Na_2SeO_3) to purified diets improved growth, decreased the incidence of neurological signs and delayed the onset of mortality in rats given up to 25 mg Hg/L as methylmercury in the drinking water (Ganther *et al.* 1972a, 1972b; Ohi *et al.* 1975a,b). The signs of mercury toxicity (e.g. tremors, loss of balance)

and mortality did not occur in chicks or quail when 8 mg Se/kg, as sodium selenite, was added to the diets containing 20 mg Hg/kg (Sell and Horani 1976). The protection becomes more evident as the acuteness of methylmercury poisoning decreases (Ohi 1975b).

In vitro experiments have shown that 0.40 mg Se/L as sodium selenate and 0.16 mg Se/L as sodium selenite have a protective effect on the nervous tissue of rat cerebella against the neurotoxicity of methylmercuric chloride (Kasuya 1976).

The evidence for the other side of selenium-mercury antagonism, i.e. whether or not mercury decreases selenium toxicity, is not clear. Ganther and Sunde (1974) quote results of animal experiments, e.g. chicks, Japanese quail, rats, in which $HgCl_2$ and methylmercury decreased the toxicity of Na_2SeO_3. Oral administration of methylmercury produced selenium deficiency in swine fed on a low-selenium diet (0.03 to 0.05 mg Se supplemented with 0.10 mg Se, as Na_2SeO_3, per kilogram of diet). Selenium deficiency signs did not occur in pigs fed the low-selenium diet without mercury nor did mercury toxicity signs occur in pigs given mercury with higher dietary selenium (the same diet as above but supplemented with 5 mg Se/kg as Na_2SeO_3) (Froseth *et al.* 1974). Results obtained by Parizek *et al.* (1974; Parizek 1976) have shown that administration of selenite or dimethyl selenide, and mercuric compounds, in this order results in a marked increase in the toxicity of the selenium compounds. Similarly, a slight synergistic effect was detected when a mixture of $Na_2SeO_4/HgCl_2$ was injected into chicken egg yolk. The hatchability was 10 to 13% lower than that predicted from the hatchability frequencies of the individual metals (Birge *et al.* 1976).

Following the finding that rat tissue contained selenium and mercury in a molar ratio of 1:1 after oral administration of $HgCl_2$ and Na_2SeO_4 and that same ratio was the most effective in counteracting selenium toxicity in chicks (Hill 1974), it has been suggested that Se reacts directly with Hg(II) and forms a stable compound (Groth *et al.* 1976). The addition of a sulphur amino acid was found to enhance the effect of selenium in detoxifying mercury, thus suggesting that the SH group is also involved in the selenium-mercury interaction (Frost and Lish 1975).

Other known physiological antagonists of selenium are copper and arsenic, and maybe lead (Schrauzer *et al.* 1977b; Frost and Lish 1975).

Copper at a level of 500 mg/kg in the diet partly counteracted the toxic effect of 40 mg Se/kg on the growth of chicks and decreased their mortality to 6.1% vs. 12.1% for the control group (no selenium and no copper) (Hill 1975a). Similar results were obtained by Jensen (1974, 1975). Copper at a concentration of 1000 mg/kg reduced mortality and partially prevented the growth depression of chicks caused by 40 to 80 mg Se/kg of diet. Large doses of copper also produced selenium deficiency. It is possible that copper acts as a selenium antagonist in a manner similar to mercury and cadmium (Schrauzer *et al.* 1977b).

Several arsenic compounds, including some organic arsenicals, protect against selenium toxicity. Administration of 5 mg As/L in the drinking water almost completely prevented the toxic effect on hatchability of 10 mg Se/kg in the diet. Similarly, 5 mg As/L in the drinking water prevented the toxic effects on the growth of chicks of 10 mg Se/L (Hill 1975a). It was shown that arsenic actually inhibited the expiration of volatile methylated selenium compounds while stimulating the fecal excretion of selenium and increasing the level of selenium in the liver and kidney (Hill 1975a). Experiments with radioarsenic have shown that selenium increased biliary excretion of arsenic just as arsenic increased biliary excretion of selenium. Several possible mechanisms have been suggested including increased permeability of the liver or the formation of a seleno-arsenic conjugate, but no definite conclusions have been reached (Levander 1971; Hill 1975a).

Tin, as $SnCl_2$, at 200 mg/kg in the diet and tellurium at 200 mg/kg as K_2TeO_2, but not as TeO_2, partially alleviated the growth depression caused by 25 mg Se/kg as H_2SeO_3 (Howell and Hill 1977). Dietary silver at 1000 mg/kg, as silver nitrate, prevented the severe growth depression and high mortality of chicks caused by high selenium (40 to 80 mg/kg) (Jensen 1974, 1975).

Oral administration of selenate prevented the toxicity of thallium but at the same time increased the retention of thallium in the liver, kidney and bones. Subcutaneous injection of thallium acetate to rats increased the retention of selenium in the liver and kidney and diminished pulmonary and urinary excretion of selenium (Rusiecki and Brzezinski 1966; Levander and Argrett 1969). Other elements—arsenic, silver, copper and tellurium, but not tin—also increased the selenium content of liver.

Data quoted by Hill (1975a) showed that the sulphate ion, which is very similar to the selenate ion, partly counteracted the growth-depressing effect of selenium, but did not change substantially the liver-diminishing effect of selenium in rats. Sulphate treatment increased urinary excretion of selenium and decreased excretion through the intestinal tract.

Studies with rats have shown that some fraction of the linseed oil meal, other than protein, and methionine fed in conjunction with vitamin E and a series of fat-soluble antioxidants, such as N, N^1-diphenyl-p-phenylene-diamine, ethoxyquin and butylated hydroxytoluene, all gave protection against liver damage caused by selenium. It has been suggested that linseed oil meal or some of its metabolites might complex with selenium and thus prevent it from poisoning sulphhydryl or other groups in the cell susceptible to selenium. The mechanism in the protection from selenium toxicity when feeding methionine, vitamin E and the fat-soluble antioxidants apparently is somewhat different. Expiration of volatile selenium compounds via the lungs or the urinary excretion of trimethyl selenide, or a combination of the two pathways, could conceivably be operating in this case (Levander 1971).

Growth retardation in chicks caused by 20 mg Se/kg in the diet was significantly decreased by the addition of 0.1% ascorbic acid to the diet (Hill 1975b).

Studies on laboratory animals have also shown that significant decreases occur in the concentrations of ascorbic acid, vitamin K, and glutathione as a result of selenium intoxication (Cooper and Glover 1974).

Aquatic Life and Wildlife

Movement of Selenium through the Aquatic Ecosystem

The transfer of selenium from terrestrial to aquatic ecosystems was shown to be slow when compared with mercury. Over 75% of the applied selenium was retained in the soil and 9% was found in the sediments. The macro-invertebrates, represented by snails, absorbed 0.033% and fish even less, 0.007%. The role played by the phyto-plankton was not studied (Huckabee and Blaylock 1974). Lake sediments seem to act as a sink for selenium.

The transport through the aquatic system has not been fully explained, though selenium is known to be methylated biologically in lake sediments (Chau *et al.* 1976). Although some of the methylated selenium metab-olites were stated to be less toxic than the soluble inorganic salts, in contrast with mercury (NAS 1976 p. 143), the whole system is not fully understood and caution must be observed, as more toxic forms of selenium may be formed, as was observed in laboratory experiments using newly hatched zebrafish. Experimental dishes had to be cleaned frequently to obtain reliable mortality data. A distinct odour characteristic of many organoselenium compounds was present, after 3 or 4 days, in the opaque film on the dishes which contained the inorganic selenium compounds. The conversion was probably due to microbial methylation of the inorganic selenium (Niimi and LeHam 1976).

The rate of uptake of selenium by phytoplankton depended on the selenium compound. More selenium was picked up from selenomethionine than from sodium selenite. Zooplankton picked up selenium directly from the selenite solution but not from selenomethionine. The latter compound was obtained from ingested phytoplankton. Transfer of selenium to fish was probably via the food. The uptake of selenium by phytoplankton was inhibited by 2,4-dinitrophenol, which uncoupled phosphorylation, and by cyanide, which prevented electron transport in the cytochrome chain. Methionine and ethionine reduced the uptake of selenomethionine, probably by competitive inhibition (Sandholm *et al.* 1973).

Selenium Levels in Fish

The concentration of selenium in a number of species of fish (fresh water and marine) had a wide range, from 0.16 to about 0.68 mg/kg wet weight. The average level in muscle from a series of fish from central Canada was 0.26 mg/kg, with most of the levels falling into the above range. Levels in fish from the Great Lakes were from 0.06 to 2.0 mg/kg (IJC 1976). Fish mortality from the accumulation of selenium through the food chain has been demonstrated (Barnhart 1958). The toxicity of the selenium content in fish eaten by wildlife is unknown, but since 3 mg Se/kg of diet has been shown to be toxic to small mammals (rats), the level of selenium in fish should be of concern.

Toxicity of Selenium

Very little information has been found on the toxicity of selenium to invertebrates and other macroscopic aquatic life. *Daphnia* sp. (Crustacea) was found to be as sensitive as *Scenedesmus* sp. (phytoplankton) with a lethal threshold of 2.5 mg/L (Bringmann and Kuhn 1959). The growth of the algae *Chlorella* sp. and *Haematococcus* sp. was reduced by 0.05 mg Se/L by 24% and 62% respectively (Hutchinson 1973).

The growth and photosynthetic activity of *Phoridium luridum* var. *olivacea* (blue-green alga) was significantly affected by 0.79 mg Se/L (10^{-5} mol/L as sodium selenite). There was no effect on photosynthesis or cell morphology at a concentration of 0.079 mg Se/L 10^{-6} mol/L as sodium selenite (Sielicki and Burnham 1973).

A number of authors have studied the effects of selenium on fish. The 96-h LC_{50} in soft water (36 mg/L as $CaCO_3$) for rainbow trout was 12.5 mg/L (as sodium selenite). A 16-day LC_{50} was 5.0 mg/L. In addition to mortalities, skin lesions not related to fungal or bacterial causes occurred on fish exposed to concentrations above 7 mg/L. Long-term studies using sodium selenite on developing eggs and hatched fish showed that the level of "no effect" lay between 0.04 and 0.08 mg Se/L (Davies and Goettl 1975).

Brook trout tested in a medium hard water (157 mg/L as $CaCO_3$) had a 96-h LC_{50} of 14.3 mg/L. The fathead minnow was less tolerant; the 96-h LC_{50} for fry was 2.9 mg/L and for juveniles, 7.3 mg/L. The size or age of the fish had an effect on its tolerance. The 168-h LC_1 (LC = lethal concentration; 1% kill) for fathead minnow juveniles was 0.85 mg Se/L as SeO_2 (Cardwell *et al* . 1976). These test fish also exhibited skin lesions as observed by Davies and Goettl (1975).

The lowest concentration of selenium that significantly impaired the conditioned response in goldfish (*Carassius auratus*), not one of the more sensitive species, was 0.25 mg/L or 1/4 of the LC_1 and 1/48 of the LC_{50}. The impairment at 0.15 mg Se/L was not significant (Weir and Hine 1970).

The effects of selenium (as SeO_2) were examined on the early life stages of zebrafish (*Brachydanio rerio*). The mortality of developing embryos was negligible at the levels tested (0.5 to 10 mg/L) and hatching was not affected, but the mortality increased sharply at 3.0 mg/L or higher when the larval fish had hatched. No effect was observed at 1.0 mg/L. Mortality caused by a specific concentration of selenium increased with longer periods of exposure; 3 to 5 mg/L gave a larval mortality of 10% in 96 h and approximately 50% at 240 h (Niimi and LaHam 1975).

Different selenium compounds had different toxicities to zebrafish. Selenium dioxide was the most toxic of the compounds tested with a 96-h LC_{50} of 20 mg Se/L. The 96-h LC_{50} values for other compounds ranged from 23 mg/L for sodium selenite to 82 mg/L for sodium selenate. Reliable figures were not obtained for the organics selenomethionine or selenocystine in the 96-h tests. Tests of longer duration to determine the concentration of selenium that would kill 5% of the fish in 10 days gave the following results: sodium and potassium selenite, 1.0 mg/L; selenium dioxide, 3.0 mg/L; potassium and calcium selenate, 17 mg/L; sodium selenate, 19 mg/L; and selenocystine, 12 mg/L. The concentration of selenomethionine was lower than the range used in the experiments, so the extrapolated concentration was 0.1 mg/L (Niimi and LaHam 1976). The concentration for selenium dioxide was similar to that obtained for the LC_1 with goldfish (Weir and Hine 1970).

A study by Goettl *et al*. (1976?) indicated that the maximum acceptable toxicant concentration (MATC) lay between 0.04 and 0.08 mg Se/L for rainbow trout (*Salmo gairdneri*).

Effect of Selenium on the Toxicity of Other Elements

Experiments with mammals have shown that arsenic counteracts chronic and acute selenium poisoning, causing an increased clearance of selenium from the liver. Selenium has been shown to be antagonistic to some heavy metals, e.g. mercury, cadmium and silver. The mechanisms ameliorating the effect of some toxicants were shown to be an alteration in the biological activity or the distribution of the metal in the tissues rather than an effect on its absorption or elimination from the body; or by the conversion to a less toxic form, e.g. methylmercury to the mercuric ion (Ganther 1974).

Selenium is antagonistic to cadmium toxicity and ameliorated its effect on the growth of the test organisms. When *Chlorella vulgaris* (green alga) was exposed to the two elements alone, cadmium was more toxic; 0.05 mg Cd/L reduced the growth to 55% and 0.05 mg Se/L reduced the growth to 70% of the control. In combination, the growth was only reduced to 79% of the control. At 0.05 mg Cd/L and 0.1 mg Se/L, the growth of *C. vulgaris* was stimulated compared to the control: *Haematococcus* sp. (green alga) showed a similar response (Hutchinson and Stokes 1975).

Mercury toxicity to chubs (*Semotilus atromaculatus*) was ameliorated by pretreatment with 3 mg Se/L for 48 hours. The pretreatment had no obvious deleterious effects on the fish over that period of time but apparently protected against mercury toxicity. The selenium did not prevent the accumulation of inorganic mercury (exposure levels 0.01, 0.04 and 0.07 mg/L) in the body but protected against its toxicity. Thirty per cent of the untreated fish with the same load of mercury died. At higher levels—0.1, 0.13 and 0.16 mg Hg/L—the body burden of mercury was lower than the untreated fish (Kim *et al*. 1977).

The opposite effect of mercury-selenium antagonism was demonstrated by Huckabee and Griffith (1974) when selenium and mercury were shown to enhance the toxic effect on carp eggs (*Cyprinus carpio*). The mechanism of

the synergistic effect was not understood, but both elements have an affinity for SH groups and may be reacting with SH groups in the egg membrane. Most studies have shown antagonism between mercury and selenium. Selenium has also been shown to be antagonistic to a number of other elements, e.g. sulphur (Weissman and Trelease 1955), thallium (Rusiecki and Brzezinski 1966) and silver (Jensen 1975).

The above examples show that care will have to be exercised in determining the safe level for selenium in natural waters, especially since inorganic selenium may be converted to organic forms, some of which are more toxic, e.g. selenomethionine, than the organic compounds containing no selenium.

Recommended Objective for Aquatic Life and Wildlife

In determining a safe level for an element in fresh water, the results of a number of studies, including long-term ones, should be taken into account.

The level of "no effect" (MATC—maximum acceptable toxicant concentration) for selenium lay between 0.04 and 0.08 mg/L (Davies and Goettl 1975) for rainbow trout. A level of 0.15 mg/L did not significantly affect the conditioned response of goldfish (Weir and Hine 1970). The mortality of larval zebrafish was not affected at levels below 3 mg Se/L (Niimi and LaHam 1975), but exposure over 10 days for adult zebrafish at concentrations of 1 mg/L selenite killed 5%. The 10-day LC_5 for selenomethionine was extrapolated to be 0.1 mg Se/L (Niimi and LaHam 1976), and was similar to the LC_1 for goldfish (Weir and Hine 1970). The growth of *Chlorella vulgaris* was reduced to 75% of the control growth by 0.05 mg Se/L.

Inorganic selenium has been shown to be converted to organic forms by biological agents and since, for example, selenomethionine was 10 times more toxic than the inorganic form, this must be taken into consideration.

The International Joint Commission (IJC 1976) did not propose a level for aquatic organisms, as the suggested level of 0.01 mg/L for raw public water supplies was considered to be stringent enough to protect them. The Environmental Protection Agency (EPA 1976) suggested 0.01 of the 96-h LC_{50} using a sensitive resident species.

The fathead minnow fry had a 96-h LC_{50} of 2.9 mg/L, and using the factor of 0.01, a safe level of 0.029 mg/L is derived. Using the lower MATC of 0.04 mg/L for rainbow trout and a 96-h LC_{50} of 12 mg/L, the application factor (MATC/96-h LC_{50}) is 0.0033. Applying this factor to the 96-h LC_{50} for other fish, e.g. 2.9 mg/L for minnows, the safe level would be 0.009 mg/L, which is very close to the level of 0.01 mg/L. The use of application factors between species must be used with caution, but they indicate the order of magnitude which should be considered.

Until further research on the toxicity of selenium to freshwater organisms is available and the fact that methylation may occur, an objective of 0.01 mg/L is recommended to protect aquatic life.

Water Supply for Livestock

Selenium occurs in all cells and tissues of the body in various concentrations depending on the tissue, animal species and the selenium level in the diet (Underwood 1971, p. 324). Positive correlations have been established between the geochemical environment and the selenium intake (Oldfield *et al*. 1974).

It has been estimated that the selenium requirement for average growth of some farm animals, e.g. chicks, pigs, calves and lambs, is of the order of 0.06 mg/kg of dry diet (Underwood 1976). Estimates of dietary requirements of livestock at 0.1 mg/kg and turkey at 0.2 mg/kg are slightly higher (NAS 1974, p. 66).

The highest selenium concentrations are found in the kidney (especially the kidney cortex), followed by glandular tissues (especially the pancreas and the pituitary), liver, etc. The muscles, bones and fatty tissues are generally low in selenium. In healthy pigs, for example, the tissue concentrations on a dry basis ranged from 0.52 mg/kg in the skeletal muscles to 11.47 mg/kg in the kidney. The liver and the pancreas contained 1.82 and 1.42 mg/kg, respectively (Lindberg 1968; Rico *et al*. 1976). On a wet tissue basis in newborn lambs, the kidney cortex contained 0.41 to 1.06 mg/kg and the liver, 0.083 to 0.350 mg/kg (Hartley 1967).

The kidney and liver levels are the most sensitive indicators of the selenium status of an animal. In sheep, concentrations greater than 1 mg/kg in the kidney cortex and 0.1 mg/kg in the liver are considered normal. Concentrations below 0.25 mg/kg (kidney cortex) and 0.02 mg/kg (liver), on a fresh basis, indicate serious selenium deficiency (Andrews *et al*. 1968). Hartley (1967) estimated that the normal selenium levels in young sheep are 1 mg/kg in the kidney cortex and 0.25 mg/kg in the liver. Allaway *et al*. (1968) estimated that a concentration of 0.21 mg/kg in the liver is near the critical minimal level for lambs.

In Holstein calves, the selenium content of tissue was not affected at 0.1 mg/kg supplemental selenium, as sodium

selenite, in the diet. Selenium levels in the blood, liver and heart were elevated significantly with 1.0 mg/kg supplemental selenium. At the same level (1 mg/kg) of dietary selenium, its retention in most other tissues was reduced significantly. Neither feed intake nor calf weight gain was affected by 0.1 to 1.0 mg/kg supplemental dietary selenium (Kincaid 1977). At toxic intakes of selenium, 10 to 100 times greater than those normally ingested, selenium concentrations in tissue rise until levels of 5 to 7 mg/kg (liver and kidney) and 1 to 2 mg/kg (muscles), on a fresh basis, are reached. Beyond these concentrations excretion begins to keep pace with absorption. Higher levels may be found in the hair and hooves of severely affected animals. The hair of yearling cattle on a seleniferous range contained as much as 30 mg/kg compared with 1 to 4 mg/kg in the hair of cattle from non-seleniferous areas (Olson *et al.* 1954).

The selenium concentration in blood changes with the selenium concentration in the diet. Normal levels in the whole blood of sheep and lambs have been reported to range between 0.06 and 0.20 mg/L (Kuchel and Buckley 1969; Hartley 1967). The selenium concentration in the blood of sheep dying from experimentally induced selenium toxicity was between 1.34 and 3.1 mg/L (Rosenfeld and Beath 1945).

Data quoted by Underwood (1971, pp. 328-329) show that the selenium concentration in cow's milk can range from approximately 0.003 mg/L in low-selenium areas to 0.067 mg/L in high-selenium areas. Selenium treatment of cows can raise these natural levels considerably. Seventy to seventy-five per cent of the selenium occurs in the protein fraction of milk. Underwood (1971, p. 329) also quoted data on selenium concentration in eggs. A normal egg contained, on average, 0.010 to 0.012 mg of selenium in a ratio of 6.3:1 between the yolk and the white parts. The average concentrations ranged between 0.056 mg/kg in low-selenium areas and 0.5 mg/kg in high-selenium areas. Toxic intakes of selenium can increase these levels 2 to 4 times.

The absorption of selenium via the gastrointestinal tract; its retention and distribution within the body; and the routes, amounts and forms of excretion depend on the animal species, chemical form, amount ingested and the presence of other elements in the diet (Underwood 1971, pp. 329-330). Experimental results showed that monogastric animals have higher intestinal absorptions than ruminants. Sheep on a diet containing 0.35 mg Se/kg had a net absorption of 35%, whereas pigs on a diet of 0.50 mg Se/kg had a net absorption of 85% (Wright and Bell 1966). The absorption of selenium from naturally seleniferous diets is somewhat higher than that from selenites and selenates. The absorption of elemental selenium or selenides is low (Underwood 1971, p. 330). It has been found that with pigs a smaller proportion of the administered dose is retained if the selenium concentration of the basal diet has fulfilled the physiological requirements of the animal (Cooper and Glover 1974). Absorbed selenium is at first carried mainly by the plasma, probably in association with plasma proteins, from which it enters all tissues, including bones and hair.

Selenium is eliminated via the feces, urine and respiratory tract. Of the selenium administered, 35 to 45% was excreted within 48 h and 50 to 60% within 10 to 12 days (Cooper and Glover 1974; Rico *et al.* 1976). Arsenic increases the excretion of selenium into the gastrointestinal tract via the bile fluid. Sulphate ion had a similar effect when selenium was administered as selenate but not as selenite (Underwood 1971, pp. 330-331).

Animal studies have shown that selenium is transmitted through the placenta with a little more difficulty in the inorganic form (Underwood 1971, p. 331).

Essentiality of Selenium to Animal Health

During the past twenty years selenium has been recognized as an essential element for several animal species. Deficiency symptoms appear at a feed dry matter level of 0.02 to 0.04 mg Se/kg or lower (Oldfield *et al.* 1974). A selenium dietary deficiency results in degenerative changes that vary with the animal species. Effects of selenium deficiency are more frequent in ruminants than in monogastric animals (Oldfield *et al.* 1974). In ruminants the effects consist mainly of myopathy, especially of the heart and skeletal muscles. Exercising a selenium-deficient animal can precipitate acute myocardial deficit and death. Periodontal disease and impaired reproduction have been reported in New Zealand sheep. Rats and swine respond to selenium deficiency with hepatic degeneration. Other organs and tissues are also affected. Selenium deficiency in poultry is associated with exudative diathesis, poor growth, poor feathering, and fibrotic degeneration of the pancreas (Oldfield *et al.* 1974).

Results quoted by Underwood (1971, p. 335) showed that selenium therapy was effective in the treatment of various myopathies, including white muscle disease (WMD) in lambs, calves, pigs, foals and chicks; dietary liver necrosis in pigs; and exudative diathesis in chicks. WMD is a degenerative disease of the striated muscles occurring at birth or in very young animals. In congenital or severe cases it leads to death, however, mildly affected animals may recover spontaneously. Among other things WMD is characterized by subnormal concentrations of selenium in the blood and tissues (Underwood 1971, p. 335ff).

Dietary liver necrosis is also characterized by a depletion of the selenium tissue concentrations and high mortality (Underwood 1971, p. 335ff). Other selenium-responsive diseases are (i) the "ill-thrift" condition occurring in lambs, beef, and dairy cattle and characterized by a sub-clinical growth deficit to rapid loss of condition and mortality, and (ii) the periodontal disease of ewes characterized by loosening and shedding of permanent molars and sometimes also of incisors, in association with gingival hyperplasia, resorption, etc. (Underwood 1971, p. 339ff). Selenium was also shown to promote growth, improve fertility and reduce postnatal losses in sheep.

The selenium requirement for prevention of pancreatic degeneration in chicks was found to be 0.01 mg/kg of diet at very high levels of vitamin E intake (100 IU/kg). At normal levels of vitamin E intake (10-15 IU/kg), 0.02 to 0.04 mg Se/kg was required (Scott 1971). Proteins containing selenium are essential components of certain bacterial and mammalian enzyme systems. Selenium-containing proteins are involved in the oxidation-reduction reactions catalyzed by formate dehydrogenase of *Escherichia coli*, glycine reductase system of *Clostridium stricklandii* and glutathione peroxidase of erythrocytes. The catalytic function of a selenoprotein isolated from the skeletal muscles of sheep has not been identified yet (Stadtman 1974; Hoekstra 1975). Glutathione peroxidase, for example, contains four selenium atoms per molecule, with an average of one selenium atom per protein subunit of about 22 000 molecular weight. The level of activity of this enzyme correlates well with the level of dietary selenium. Its activity is reduced in the tissues of selenium-deficient animals. From experimental studies with chicks, ewes and lambs, it seems that the determination of the level of glutathione peroxidase in blood plasma or red cells provides a sensitive index of dietary selenium imbalance (Underwood 1976).

Selenium deficiency significantly increased the toxicity of the herbicide paraquat to mice from an LC_{50} value of 30 mg per kilogram of animal chow diet for the control group to 10.4 mg/kg in the selenium-deficient group. Liver glutathione peroxidase activity in the selenium-deficient mice was 16% of the control activity (Bus *et al.* 1975). A selenium addition (as selenite) of 3.75 mg/kg in the diet of albino rats improved litter production and survival, and the growth of postweaning male animals was stimulated by a 0.1 to 1.0 mg/kg addition (Halverson 1974).

Selenium Toxicity to Animals

Acute selenosis, called blind staggers, usually results from consumption of accumulator plants, some of which contain as much as 1000 mg Se/kg. The animals become blind and often paralyzed. Death usually occurs as a result of starvation and respiratory failure. Excessive intake of selenium, usually by the consumption of normal forage by grazing animals in seleniferous areas, might lead to naturally occurring chronic selenosis called also "alkali disease". It is characterized by dull, rough hair coat, loss of hair from the mane and tail (horses), sloughing of hooves, dullness and lack of vitality, stiffness and lameness because of erosion of joints, liver cirrhosis and anemia (Johnson 1976). Seleniferous diets might result in an unusually high incidence of birth defects (Horvath 1976). Newborn foals and calves, for example, might have deformed hooves.

Levels of 2 to 3 mg Se/L, as selenate or selenite, in the drinking water of rats caused a small decrease in their weight in 4 to 6 weeks. At a level of 6 to 9 mg/L considerable mortality occurred (Palmer and Olson 1974). The symptoms of "alkali disease" appeared in cattle which were fed a diet containing 15 mg Se/kg of diet (wet basis), as selenite (Olson and Embry 1973). Young pigs fed selenium, as sodium selenite or selenomethionine, at concentrations of 20 to 600 mg/kg had emesis, anorexia, loss of weight, cachexia, central nervous system depression, respiratory distress and coma, along with subnormal body temperature and death. The lesions of selenosis included fatty metamorphosis and centrilobular necrosis in the liver, congestion of the renal medulla, necrosis in lymphoid follicles, edema and degenerative changes in the cerebrum, cerebellum and spinal cord. Lesions of the central nervous system were more common in swine given inorganic selenium, and pancreatic lesions were associated with pigs given organic selenium. The estimated mean lethal dose based on the amount of selenium fed was 9.4 mg/kg of body weight in pigs fed sodium selenite and 13.6 mg/kg of body weight in pigs fed selenomethionine (Herigstad *et al.* 1973). Horvath (1976) concluded that toxicity begins at a dietary selenium concentration of approximately 5 mg/kg. Selenates and selenites are more toxic than elemental selenium or selenides. Low-protein, low-sulphate diets increase the toxicity of selenium.

Sodium selenate injected into the egg's yolk to give a concentration of 0.001 mg/kg reduced the hatchability to 64 to 66% of that of the control group. The TL_{50} value was approximately 0.01 mg/kg. There was an appreciable percentage of defective birds among the survivors at or above TL_{50} (Birge and Roberts 1976). Egg size and percentage of hatchability decreased when 8 mg Se/kg, as sodium selenite, was added to the diet of laying chickens (Arnold *et al.* 1973). In another study dietary levels of 2 mg Se/kg and above depressed hatchability, and the incidence of embryo abnormalities and malpositions was high (Fitzsimmons *et al.* 1975).

Recommended Objective for Water Supply for Livestock

Selenium is both essential and toxic to animal life. The margin of safety between requirement and toxicity is about fifty fold. The minimum selenium concentration recommended for foodstuff was 0.1 mg/kg (Green 1972). The toxic levels seem to start at a concentration of approximately 5 mg/kg.

The National Academy of Sciences in the United States (NAS 1973, p. 316) has recommended that the upper limit for selenium in livestock waters be 0.05 mg/L. The Australian Water Quality Criteria (Hart 1974, pp. 126-127) has recommended a working level for selenium in stock drinking waters of 0.02 mg/L. Both levels are below the minimum concentration recommended for foodstuffs. Considering, however, the large volumes of water consumed by livestock, e.g. beef cattle 26 to 66 L/d, dairy cattle 38 to 110 L/d, horses 30 to 45 L/d, swine 11 to 19 L/d (adult animals of medium height, in a temperate climate) (NAS 1974, p. 27), and the fact that selenium in water is present as selenate or selenite and it is easily absorbable, a maximum selenium concentration of 0.05 mg/L is recommended for livestock water supplies.

Irrigation Waters

Selenium in Plants

The selenium content of plants varies over a wide range depending mainly on the type of plant. Other factors involved are the selenium content of the soil, the chemical form of selenium, and the weather (Moxon and Olson 1974).

One important group of plants with respect to selenium are the so-called "indicator plants". They grow on soils containing high levels of selenium and are therefore helpful in locating seleniferous soils. From the point of view of selenium accumulation, plants can be divided into three groups (Rosenfeld and Beath 1964).

(i) Primary indicators. They require selenium for their growth, and they accumulate selenium at very high levels, often as much as several grams per kilogram.

(ii) Secondary indicators. They do not appear to require selenium for their growth, but they accumulate the element when grown on soils with high selenium content.

(iii) Normal plants, which do not normally accumulate selenium in excess of 50 mg/kg (dry weight).

The indicator plants sometimes are also referred to as "converter" plants because they appear to extract selenium from soil in a form that is not available to other plants, redepositing it in a form that is available. The exact mechanism of this "conversion" is not known, but one possible explanation is that many of the "indicator" plants are deeply rooted, therefore they may remove soluble selenium from the subsoil and deposit it on the surface where it is more accessible to other plants (Moxon and Olson 1974).

Accumulation of the selenium analogs of methionine, S-methylcysteine, gamma-glutamylcysteine and cystathionine appears to account, at least partially, for the high selenium content of some species of selenium indicator plants (Stadtman 1974).

Data quoted by Johnson *et al* (1967) on plant uptake and distribution of selenium from artificially selenified soil under greenhouse conditions showed that accumulator plants grown on soil containing 20 mg Se/kg, as selenate or organic selenium, had concentrations between 304 mg/kg in western aster and 8078 mg/kg in tansy aster. All range plants, including timothy and alfalfa, accumulated selenium. Forage with alfalfa contained 40 mg Se/kg at the 5 mg/kg selenate treatment of soil and 33 mg/kg with timothy at 3 mg/kg selenate treatment of soil.

Crop plants also accumulated selenium. For example, in soil treated with 0.1 mg Se/kg (as selenate) the concentration in the grain (dry basis) ranged from 2.5 mg/kg for barley to 8.9 mg/kg for wheat. The straw part had lower concentrations: 1.0 and 5.7 mg/kg, respectively. A suggestion that selenium distribution in plants is related to the distribution of protein (Moxon and Olson 1974) is not always supported by experimental data, since when no selenium was added to the soil, for some crops—barley, oats, and rye—the straw part of the plant had a higher concentration than the grain on a dry matter basis (Gissel-Nielsen and Bisbjerg 1974). Similarly, in soil treated with 3 mg Se/kg (as selenate) the concentration (dry basis) in the grain was between 15 mg/kg in oats and 81 mg/kg in wheat. The concentration (dry basis) in the straw part ranged between 18 mg/kg in oats and 112 mg/kg in wheat. Treatment with 20 mg/kg organic selenium (from powdered *Astragalus* plants, an indicator plant) resulted in higher concentrations in the plants: 10 mg/kg in the grains of safflower to 111 mg/kg in the wheat grains and from 27 mg/kg in the straw of oat plants to 151 mg/kg in the wheat straw. At the same treatment sunflower plants accumulated very high amounts: 392 mg/kg (Hamilton and Beath 1963).

At identical selenium concentrations in soil, there was only a small difference between selenium concentrations in plants grown on soil treated with selenium as selenate and organic selenium. All plants were capable of converting inorganic selenium to organic and vice versa (Hamilton and Beath 1963).

Vegetables grown in greenhouses on soils containing either 3 mg Se/kg (as selenate) or 20 mg/kg organic selenium absorbed, metabolized and stored variable amounts of selenium in their tissues. The amount stored in the edible parts, on a fresh basis, varied from 12 mg/kg in cucumber to 80 mg/kg in pea seed. On a dry basis cucumber again had the lowest concentration, 18 mg/kg, and cabbage the highest, 159.7 mg/kg, most of it in water-soluble organic and inorganic forms. The inedible parts of the vegetables, with only a few exceptions (broccoli, radish), contained much higher levels of selenium than the edible portion. On a dry basis, the selenium concentration in the inedible portions of a series of common vegetables ranged from 27 mg/kg in potato peel to 378 mg/kg in cabbage stem. On a fresh basis, the respective concentrations were 7.0 and 91.5 mg/kg (Hamilton and Beath 1964). All vegetable plants possessed the ability to convert a portion of the absorbed selenium as selenate into an organic form, and in most of the vegetables studied, selenium uptake was greater when they were grown on soils containing organic selenium. It was not clear, however, how this increased uptake related to the higher selenium concentration in the soils treated with organic selenium. In all instances, the soil selenium levels at the end of the experimental period were nearly equal, less than 0.5 mg/kg lower than the levels present at the beginning.

Mushrooms of the genus *Agaricus* also accumulated selenium from soil. Thus, *A. edulis* grown on soil containing 0.052 mg Se/kg contained 3.8 to 7.8 mg/kg (dry weight) in the carpophores. This represented an accumulation factor of 75 to 150 (Stijve and Besson 1976).

In general the selenium contents of soils and plants do not correlate well. On the other hand, apparently the water-soluble selenium in alkaline soils is a fairly good index of the availability of the element to the plants if the oxidation state of selenium is the same (Moxon and Olson 1974). Water-soluble selenium is probably present as selenate in alkaline soils. In acidic or neutral soils selenium from selenite is precipitated by ferric hydroxide, forming adsorption complexes rather than crystalline ferric selenite (Geering *et al.* 1968). Laboratory experiments with pure systems indicated that the rates of conversion of SeO_3^{2-} to SeO_4^{2-} and vice versa are sluggish, whereas the transformation of SeO_3^{2-} to Se^0 proceeded very rapidly. The oxidation of Se^0 to SeO_3^{2-} is very difficult to effect unless Se^0 is finely dispersed. In natural systems, however, it is possible that certain agents, such as bacteria, in the soil assist the conversion of selenium to more soluble forms.

Gissel-Nielsen and Bisbjerg (1970) have shown that plants pick up very little elemental selenium from soil (0.01%) and that selenates are picked up much more readily than selenites, leading to concentrations up to approximately 50 times greater in plants. The amount picked up by plants is nearly the same regardless of the solubility of the selenates or selenites involved. In field experiments, in two years plants removed about 10 to 30% of the selenate and 1 to 4% of the selenites added at an initial concentration of 0.1 mg/kg (or 250 g selenium per hectare).

Recommended Objective for Irrigation Waters

The accumulation of selenium by plants depends on the plant species, the type of soil, the soil pH, the oxidation state of selenium, the concentration of selenium in soil, and the presence of other compounds such as sulphates or cadmium (Ganje 1966; Francis and Rush 1973). Selenium does not seem to concentrate in soil. In most cases, compounds which are most available to plants, such as selenates, seem to be leached away by irrigation water, runoff waters, etc. Most of the seleniferous soils are of natural origin rather than a result of human activities. Plants do not seem to be affected by the presence of selenium in soil unless the concentration is high. Although no exact value has been established for this threshold concentration, it seems to be greater than 30 mg/kg. The normal soil concentration is below 2 mg Se/kg. Some specific symptoms of selenium excess have been summarized by Ganje (1966).

When considering an objective for selenium concentration in irrigation waters, the limiting factor is the accumulation of selenium by plants used in animal or human nutrition. As mentioned before, when selenium is present in the most available form, i.e. as selenate or organic selenium, the accumulation factor of selenium by certain plants has a value of 20 to 50. It has also been shown that for human and animal nutrition, the minimum selenium concentration to protect the organism from selenium deficiency is around 0.1 to 0.2 mg/kg, and some selenium toxicity symptoms start to appear at concentrations above 4 mg/kg of diet.

Aiming at a maximum concentration in plants of 1 mg/kg, using an accumulation factor of 50, and assuming that the retention of selenium present in the irrigation waters by the soil is minimal, the recommended maximum limit for selenium concentration in these waters is 0.02 mg/L for

continuous use. For intermittent use the recommended maximum selenium concentration in irrigation waters is 0.05 mg/L. The Australian water quality criteria (Hart 1974, p. 110), and the United States National Academy of Sciences (NAS 1973, p. 345) have recommended a limit of 0.02 mg/L for selenium in irrigation waters.

Recreation and Aesthetics

Recreation waters should meet the same selenium requirement as that recommended for surface waters that are used as sources of drinking water, i.e. a maximum of 0.05 mg Se/L.

There is a possibility that fish in areas with high selenium levels in the sediments may have accumulated sufficient selenium to be a possible hazard if consumed, as the difference between optimal and toxic intake levels in food is comparatively narrow. Although the normal human intake is balanced by excretion, the consumption of contaminated fish or wildlife may have to be considered in some parts of Canada. No reports of such problems have been found to date.

Recommended Objective for Recreational Waters

The concentration of selenium in water should not exceed 0.05 mg/L total selenium.

Industrial Water Supplies

Water quality requirements for industry vary widely, but most industries are capable of treating raw water to attain a quality that is acceptable to the industry in question. With respect to the level of selenium, the industries concerned would be those involved in food and drink processing.

Recommended Objective for Industry

Water incorporated into the final product or used in the canning industry should meet the same requirements as for drinking water. A total selenium concentration of 0.05 mg/L is recommended where water undergoes a simple treatment before use. For water undergoing advanced treatment, a total selenium concentration between 0.10 and 0.25 mg/L is acceptable.

REFERENCES

Allaway, W.H. 1975. Soil and plant aspects of the cycling of chromium, molybdenum and selenium. *In* International Conference on Heavy Metals in the Environment, Symposium Proceedings, Vol. 1, Toronto, Ontario. pp. 35-47.

Allaway, W.H., J. Kubota, F. Losee and M. Roth. 1968. Arch. Environ. Health, 16:342. *Quoted in* Cooper, W. Charles and J.R. Glover 1974, p. 661.

Allaway, W.H., Herbert A. Laitinen, Hubert W. Lakin and O.H. Muth. 1974. Selenium. *In* Geochemistry and the Environment, Volume 1. National Academy of Sciences, Washington, D.C., 1974. pp. 57-63.

Allaway, W.H., D.P. Moore, J.E. Oldfield and O.H. Muth. 1966. Movement of physiological levels of selenium from soils through plants to animals. J. Nutr. 88:411.

Andrews, H.D., W.J. Hartley and A.B. Grant. 1968. N.Z. Vet. J. 16:3. *Quoted in* Underwood, E.J. 1971, p. 325.

Argo, David G. and Gordon L. Culp. 1972. Heavy metals removal in wastewater treatment processes: Part 2 - pilot plant operation. Water & Sewage Works, 119(9):128-132.

Arnold, R.L., O.E. Olson and C.W. Carlson. 1973. Dietary selenium and arsenic additions and their effects on tissue and egg selenium. Poult. Sci. 52:847-854.

Bagnall, K.W. 1973. Selenium, tellurium, and polonium. *In* Comprehensive Inorganic Chemistry, Vol. II. Edited by J.C. Bailar, H.J. Emeléus, Sir Ronald Nyholm and A.F. Trotman-Dickenson. Pergamon Press. pp. 935-1008.

Barnhart, R.A. 1958. Chemical factors affecting the survival of game fish in a western Colorado reservoir. Colorado Co-operative Fisheries Research Unit, Quarterly Report, 4:25-28. (Cited from Sport Fishery Abstracts, 1958 3(1673):114) *Quoted in* IJC 1976.

Beath, O.A. 1962. Sci. Newsletter, 81:254. *Quoted in* Cooper, W. Charles and J.R. Glover 1974.

Bertine, K.K. and Edward D. Goldberg. 1971. Fossil fuel combustion and the major sedimentary cycle. Science, 173:233-235.

Birge, Wesley J. and Oliver W. Roberts. 1976. Toxicity of metals to chick embryos. Bull. Environ. Contamin. Toxicol. 16(3):319-324.

Birge, Wesley J., Oliver W. Roberts and Jeffrey A. Black. 1976. Toxicity of metal mixtures to chick embryos. Bull. Environ. Contamin. Toxicol. 16(3):314-318.

Bowen, H.J.M. 1966. Trace Elements in Biochemistry. Academic Press, London and New York.

Brinckman, F.E. and W.P. Iverson. 1975. Chemical and bacterial cycling of heavy metals in the estuarine system. *In* Marine Chemistry in the Coastal Environment. Edited by Thomas M. Church. American Chemical Society Symposium Series 18, Washington, D.C. pp. 319-342.

Bringmann, G. and R. Kuhn. 1959. Vergleichende wasser-toxikologische Untersuchungen an Bakterien, Algen und Kleinkrebsen. Gesundheits-Ing. 80:115-120. (In German).

Brown, D.G., R.F. Burk, R.J. Seely and K.W. Kiker. 1972. Effect of dietary selenium on the gastrointestinal absorption of $^{75}SeO_3$ in the rat. Int. J. Vit. Nutr. 42:588-591. *Quoted in* NAS 1976, p. 52.

Buhler, Donald R. 1972. Environmental contamination by toxic metals. *In* Heavy Metals in the Environment. Water Resources Research Institute. Oregon State University.

Burk, R.F., Jr., W.N. Pearson, R.P. Wood II and F. Viteri. 1967. Blood-selenium, levels and *in vitro* red blood cell uptake of ^{75}Se in kwashiorkor. Am. J. Clin. Nutr. 20:723-733. *Quoted in* NAS 1976, p. 93.

Bus, J.S., S.Z. Cagen, S.D. Aust, and J.E. Gibson. 1975. Lipid peroxidation: a possible mechanism for paraquat toxicity. Toxicol. Appl. Pharmacol. 33(1):197-198.

Cardwell, R.D., D.G. Foreman, T.R. Payne and D.J. Wilbur. 1976. Acute toxicity of selenium dioxide to freshwater fishes. Arch. Environ. Contamin. Toxicol. 4(2):129-144.

Chau, Y.K., P.T.S. Wong, B.A. Silverberg, P.L. Luxon and G.A. Bengert. 1976. Methylation of selenium in the aquatic environment. Science, 192(4244):1130-1131.

Cohen, Jesse M. 1977. Trace metal removal by wastewater treatment. Technology Transfer, U.S. Environmental Protection Agency, January 1977.

Cooper, W. Charles and J.R. Glover. 1974. The toxicity of selenium and its compounds. *In* Selenium. Edited by Ralph A. Zingaro and W. Charles Cooper. Van Nostrand Reinhold Company, New York. pp. 654-674.

CRC 1970. Handbook of Chemistry and Physics, 51st Edition. The Chemical Rubber Company, Cleveland, Ohio.

Davies, P.H. and J.P. Goettl. 1975. Study of the effects of the metallic ions on fish and aquatic organisms. Water Pollution Studies N.P. Colorado Dept. Nat. Resource Div. Wildlife pp. 7-29.

Demayo, Adrian, Edward Brien and Kenneth W. Taylor. 1979. Elemental composition of fossil fuels and their ashes. A review. Unpublished report. Water Quality Branch, Department of the Environment, Ottawa.

Dickson, R.C. and R.H. Tomlinson. 1967. Clin. Chem. Acta, 19:311. *Quoted in* Cooper, W. Charles and J.R. Glover 1974, p. 661.

Diplock, Anthony T. 1976. Metabolic aspects of selenium action and toxicity. Chemical Rubber Co. Rev. Toxicol. 4(3):271-329.

Duce, Robert A., Gerald L. Hoffman and William H. Zoller. 1975. Atmospheric trace metals at remote Northern and Southern hemisphere sites: pollution or natural? Science, 187(4171):59-61.

Dutkiewicz, T., B. Dutkiewicz and I. Balcerska. 1972. Dynamics of organ and tissue distribution of selenium after intragastric and dermal administration of sodium selenite. Bromatol. Chem. Toksykol. 4:475-481. *Quoted in* NAS 1976, p. 52.

EPA. 1976. Quality Criteria for Water. U.S. Environmental Protection Agency. EPA-440/9-76-023.

Eriksson, Erik. 1977. Water chemistry and water quality. Ambio, 6(1):27-30.

Fitzsimmons, R.C., K. Phalaraksh and D.B. Bragg. 1975. Chick embryo development as influenced by selenium in the egg. Poult. Sci. 54(5):1762.

Francis, C.W. and S.G. Rush. 1973. Factors affecting uptake and distribution of cadmium in plants. *In* Trace Substances in Environmental Health-VII. Proceedings of the 7th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia. Edited by Delbert D. Hemphill. pp. 75-81.

Froseth, John A., Richard C. Piper and James R. Carlson. 1974. Relationship of dietary selenium and oral methyl mercury to blood and tissue selenium and mercury concentrations and deficiency - toxicity signs in swine. Fed. Proc. 33(3 part 1):660.

Frost, Douglas V. and Paul M. Lish. 1975. Selenium in biology. Annu. Rev. Pharmacol. 15:259-284.

Ganje, Tony J. 1966. Selenium. *In* Diagnostic Criteria for Plants and Soils. Edited by H.D. Chapman. University of California, Division of Agricultural Sci., pp. 394-404.

Ganther, Howard E. 1974. Biochemistry of selenium. *In* Selenium. Edited by Ralph A. Zingaro and W. Charles Cooper. Van Nostrand Reinhold Company, New York. pp. 546-614.

Ganther, H.E. and M.L. Sunde. 1974. Effect of tuna fish and selenium on the toxicity of methylmercury: a progress report. J. Food Sci. 39(1):1-5.

Ganther, H.E., C. Goudie, M.L. Sunde, M.J. Kopecky, P. Wagner, Sang-Huan Oh and W.G. Hoekstra. 1972a. Selenium: Relation to decreased toxicity of methylmercury added to diets containing tuna. Science, 175:1122-1124.

Ganther, H.E., P.A. Wagner, M.L. Sunde and W.G. Hoekstra. 1972b. Protective effects of selenium against heavy metal toxicities. *In* Trace Substances in Environmental Health—VI. Proceedings of the 7th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia. Edited by Delbert D. Hemphill. pp. 247ff.

Geering, Harold R., Earle E. Cary, L.H.P. Jones and W.H. Allaway. 1968. Solubility and redox criteria for the possible forms of selenium in soils. Soil Sci. Soc. Am. Proc. 32(1):35-40.

Gissel-Nielsen, G. and B. Bisbjerg. 1970. The uptake of applied selenium by agricultural plants. 2. The utilization of various selenium compounds. Plant Soil, 32(2):382-396.

Goettl, John P. Jr., Patrick H. Davies and James R. Sinley. 1976? Water pollution studies. Colorado Fisheries Research Review 1972-1975 Rev. No. 8. Fisheries Research Section. Colorado Div. Wildlife pp. 68-75.

Green, James D. 1972. Chemical form and biological availability of trace elements. Hog Farm Manage. 9(6):19-21.

Groth, David H., Lloyd Stettler and George Mackay. 1976. Interactions of mercury, cadmium, selenium, tellurium, arsenic and beryllium. *In* Effects and Dose - Response Relationships of Toxic Metals. Edited by G.F. Nordberg. Elsevier Scientific Publishing Company, Amsterdam. pp. 527-543.

Hadjimarkos, D.M. 1973. Trace elements and dental health. *In* Trace Substances in Environmental Health—VII. Proceedings of the 7th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia. Edited by Delbert D. Hemphill. pp. 25-30.

Halverson, A.W. 1974. Growth and reproduction with rats fed selenite-Se. *In* Trace Element Metabolism in Animals - 2. Edited by W.G. Hoekstra, J.W. Suttie, H.E. Ganther, and W. Mertz. University Park Press, Baltimore. pp. 587-589.

Hamilton, J.W. and O.A. Beath. 1963. Selenium uptake and conversion by certain crop plants. Agron.J. 55(6):528-531.

Hamilton, John W. and O.A. Beath. 1964. Amount and chemical form of selenium in vegetable plants. Agr. Food Chem. 12(4): 371-374.

Harr, J.R. and O.H. Muth. Selenium poisoning in domestic animals and its relationship to man. Clin. Toxicol. 5:175-186. *Quoted in* NAS 1976, pp. 114.

Hart, Barry T. 1974. A Compilation of Australian Water Quality Criteria. Australian Water Resources Council Technical Paper No. 7, Canberra.

Hartley, W.J. 1967. Levels of selenium in animal tissues and methods of selenium administration. *In* Selenium in Biomedicine. Edited by O.H. Muth, J.E. Oldfield, and P.H. Weswig. The AVI Publishing Company, Inc. Westport, Connecticut, pp. 79-96.

Hashimoto, Yoshikazu, Jae Y. Hwang and Saburo Yanagisawa. 1970. Possible source of atmospheric pollution of selenium. Environ. Sci. Technol. 4:157-158.

Herigstad, R.R., C.K. Whitehair and O.E. Olson. 1973. Inorganic and organic selenium toxicosis in young swine: comparison of pathologic changes with those in swine with vitamin E - selenium deficiency. Am. J. Vet. Res. 34(10):1227-1238.

Higgs, D.J., V.C. Morris and O.A. Levander. 1972. Effect of cooking on selenium content of foods. J. Agric. Food Chem. 20:678. *Quoted in* NAS 1966, p. 19.

Hill, C.H. 1974. Reversal of selenium toxicity of chicks by mercury, copper, and cadmium. J. Nutr. 104:593-598.

Hill, C.H. 1975a. Interrelationships of selenium with other trace elements. Fed. Proc. 34(11):2096-2100.

Hill, C.H. 1975b. The reduction of metal toxicities by ascorbic acid in the chick. Fed. Proc. 34(3):926.

Hoekstra, William G. 1975. Biochemical function of selenium and its relation to vitamin E. Fed. Proc. 34:2083-2089.

Horvath, Donald J. 1976. Trace elements and health. *In* Trace Substances and Health. A Handbook, Part 1. Edited by Paul M. Newberne. Marcel Dekker, Inc., New York and Basel, pp. 319-356.

Howell, Gary and C.H. Hill. 1977. Effects of arsenic, tin, tellurium, and lead on selenium toxicity in chicks. Fed. Proc. 36(3):1094.

Huckabee, J.W. and B.G. Blaylock. 1974. Microcosm studies on the transfer of Hg, Cd, and Se from terrestrial to aquatic ecosystems.

In Trace Substances in Environmental Health—VIII. Proceedings of the 8th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia. Edited by Delbert D. Hemphill. pp. 219-222.

Huckabee, J.W. and N.A. Griffith. 1974. Toxicity of mercury and selenium to the eggs of carp (*Cyprinus carpio*). Trans. Am. Fish. Soc. 103(4):822-825.

Hutchinson, T.C. 1973. Comparative studies of the toxicity of heavy metals to phytoplankton and their synergistic interactions. Water Pollut. Res. Can. 8:68-90.

Hutchinson, T.C. and P.M. Stokes. 1975. Heavy metal toxicity and algal bioassays. Water Quality Parameters. Am. Soc. Test Mater. Spec. Tech. Publ. 573. pp. 320-343.

IJC. 1976. International Joint Commission. Great Lakes Water Quality 1975. Appendix A. Water Quality Objectives Subcommittee Report. pp. 66-74.

Jensen, L.S. 1974. Interactions of silver and copper with selenium in chicks. Fed. Proc. 33(3, Part 1):694.

Jensen, Leo. S. 1975. Modification of a selenium toxicity in chicks by dietary silver and copper. J. Nutr. 105:769-775.

Johnson, C.M. 1976. Selenium in the environment. Residue Rev. 62:101-130.

Johnson, C.M., C.J. Asher and T.C. Broyer. 1967. Distribution of selenium in plants. *In* Selenium in Biomedicine. The AVI Publishing Company, Inc. Westport, Connecticut. pp. 57-75.

Kasuya, Minoru. 1976. Effect of selenium on the toxicity of methylmercury on nervous tissue in culture. Toxicol. Appl. Pharmacol. 35:11-20.

Kharkar, D.P., K.K. Turekian and K.K. Bertine. 1968. Stream supply of dissolved silver, molybdenum, antimony, selenium, chromium, cobalt, rubidium, and cesium to the oceans. Geochim. Cosmochim Acta, 32(3):285-298.

Kim, Jong Hwan, Eric Birks and J.F. Heisinger. 1977. Protective action of selenium against mercury in northern creek chubs. Bull. Environ. Contamin. Toxicol. 17(2):132-136.

Kincaid, Ronald Lee. 1977. Zinc and selenium metabolism in animals. Dissert. Abstr. 37(78):3288-3289.

Kubota, Joe, E.E. Cary and Gunnar Gissel-Nielsen. 1975. Selenium in rainwater of the United States and Denmark. *In* Trace Substances in Environmental Health—IX. Proceedings of the 9th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia. Edited by Delbert D. Hemphill. pp. 123-130.

Klein, David H., Anders W. Andren and Navell E. Bolton. 1975. Trace element discharges from coal combustion for power production. Water, Air, Soil Pollut. 5(1):71-77.

Kuchel, R.E. and R.A. Buckley. 1969. Aust. J. Agr. Res. 20:1099 *Quoted in* Underwood, E.J. 1971, p. 327.

Lakin, Hubert W. 1973. Selenium in our environment. *In* Trace Elements in the Environment. Edited by E.L. Kothny. Advances in Chemistry Series No. 123, American Chemical Society, Washington, D.C.

Levander, O.A. 1971. Factors that modify the toxicity of selenium. *In* New Trace Elements in Nutrition. Edited by Walter Mertz and W.E. Cornatzer. Marcel Dekker Inc., New York. pp. 57-83.

Levander, Orville A. 1975. Selenium and chromium in human nutrition. J. Am. Dietetic Assoc. 66(4):338-344.

Levander, O.A. and L.C. Argrett. 1969. Effect of arsenic, mercury, thallium and lead on selenium metabolism in rats. Toxicol. Appl. Pharmacol. 14:308. *Quoted in* Diplock, Anthony T. 1976.

Lindberg, P. 1968. Acta Vet. Scand. Suppl. 23. *Quoted in* Underwood, E.J. 1971, p. 326.

Linstedt, K. Daniel, Carl P. Houck and John T. O'Connor. 1971. Trace element removals in advanced wastewater treatment processes. J. Water Pollut. Contr. Fed. 43(7):1507-1513.

Ludwig, T.G. and B.G. Bibby. 1969. Geographic variations in the prevalence of dental caries in the United States of America. Caries Res. 3(1):32-43.

Marjanen, H. and S. Soini. 1972. Possible causal relationship between nutritional imbalances, especially manganese deficiency and susceptibility to cancer, in Finland. Ann. Agric. Fenn. 11:391-406. *Quoted in* NAS 1976, p. 94.

McMullen, Michael K. 1976. Coal and coke. *In* Canadian Minerals Yearbook 1975. Published by the Department of Energy, Mines and Resources, Ottawa.

Merritt, W.F. 1976. Trace element content of precipitation in a remote area. *In* Measurement, Detection and Control of Environmental Pollutants. International Atomic Energy Agency, Vienna, STI/PUB/432. pp. 75-87.

Money, D.F.L. 1970-71. *Quoted in* NAS 1976, p. 93.

Moxon, Alvin L. and Oscar E. Olson. 1974. Selenium in agriculture. *In* Selenium. Edited by Ralph A. Zingaro and W. Charles Cooper. Van Nostrand Reinhold Company, New York. pp. 675-707.

NAS. 1973. Water Quality Criteria 1972. National Academy of Sciences, Washington, D.C. EPA-R3-73-033.

NAS. 1974. Nutrients and toxic substances in water for livestock and poultry. National Academy of Sciences, Washington, D.C.

NAS. 1976. Selenium. Medical and Biological Effects of Environmental Pollutants. Division of Medical Sciences, National Academy of Sciences, Washington, D.C. Edited by Delbert D. Hemphill.

Neri, L.C. 1976. Some data from Canada. *In* Hardness of Drinking Water and Public Health. Proceedings of the European Scientific Colloquium. Luxembourg, May 1975. Edited by R. Amavis, W.J. Hunter, and J.G.P.M. Smeets. Pergamon Press. pp. 343-347.

NHW. 1968. Canadian Drinking Water Standards and Objectives. Department of National Health and Welfare, Ottawa.

Niimi, A.J. and Q.N. LaHam. 1975. Selenium toxicity on the early life stages of zebrafish (*Brachydanio rerio*). J. Fish. Res. Board Can. 32:803-806.

Niimi, A.J. and Q.N. Laham. 1976. Relative toxicity of organic and inorganic compounds of selenium to newly hatched zebrafish (*Brachydanio rerio*). Can. J. Zool. 54(4):501-509.

Ohi, Gen, Susumu Nishigaki, Hironobu Seki, Yukihiro Tamura, Toshio Maki, Hiroko Maeda, Setsuko Ochiai, Hiroshi Yamada, Yasuhiro Shimamura and Hiroshi Yagyu. 1975a. Interaction of dietary methylmercury and selenium on accumulation and retention of these substances in rat organs. Toxicol. Appl. Pharmacol. 32(3):527-533.

Ohi, Gen, Hironobu Seki, Hiroko Maeda and Hiroshi Yagyu. 1975b. Protective effect of selenite against methylmercury toxicity: observations concerning time, dose and route factors in the development of selenium attenuation. Ind. Health, 13(4):93-99.

Oldfield, J.E., W.H. Allaway, H.H. Draper, D.V. Frost, L.S. Jensen, M.L. Scott and P.L. Wright. 1971. Selenium in Nutrition. National Academy of Sciences, Washington, D.C. *Quoted in* Levander, Orville A. 1975.

Oldfield, James E., William H. Allaway, Herbert A. Laitinen, Hubert W. Lakin and O.H. Muth. 1974. Selenium. *In* Geochemistry and the Environment. Vol. 1. The Relation of Selected Trace Elements to Health and Disease. National Academy of Sciences, Washington, D.C. pp. 57-63.

Olson, O.E. and L.B. Embry. 1973. Chronic selenite toxicity in cattle. Proc. S.D. Acad. Sci. 52:50-58.

Olson, O.E., C.A. Denkel and L.D. Kamstra. 1954. S. Dak. Farm Home Res. 6:12. *Quoted in* Underwood, E.J. 1971, p. 325.

Palmer, I.S. and O.E. Olson. 1974. Relative toxicities of selenite and selenate in the drinking water of rats. J. Nutr. 104:306-314.

Parizek, J. 1976. Interrelationships among trace elements. *In* Effects and Dose—Response Relationships of Toxic Metals. Edited by

G.F. Nordberg. Elsevier Scientific Publishing Company, Amsterdam. pp. 498-510.

Parizek, J., J. Kalouskova, A. Babicky, J. Benes and L. Pavlik. 1974. Interaction of selenium with mercury, cadmium and other toxic metals. *In* Trace Element Metabolism in Animals–2. Edited by W.G. Hoekstra, J.W. Suttie, H.E. Ganther and W. Mertz. University Park Press, Baltimore. pp. 119-131.

Perry, H.M., Jr., E.F. Perry and M.W. Erlanger. 1974. Reversal of cadmium-induced hypertension by selenium or hard water. *Quoted in* Shamberger, Raymond J., Stanley A. Tytko, and Charles E. Willis 1975.

Pletnikova, I.P. 1970. Biological effect and safe concentration of selenium in drinking water. Hyg. Sanit. 35(2):176-181.

Rail, Chester D. and William M. Hadley. 1976. Selenium in water. An overview. J. Environ. Health, 39(3):173-175.

Rhead, W.J., E.E. Cary, W.H. Allaway, S.L. Saltzstein and G.N. Schrauzer. 1972. The vitamin E and selenium status of infants and sudden infant death syndrome. Bioinorg. Chem. 1:289-294. *Quoted in* NAS 1976, p. 93.

Rico, A.G., P. Benard and J.P. Braun. 1976. Contribution à l'étude métabolique de l'ion sélénite marqué au ^{75}Se chez différents espèces animales. *In* Nuclear Techniques in Animal Production and Health. International Atomic Energy Agency, Vienna. pp. 207-218.

Rosenfeld, I. and O.A. Beath. 1945. J. Nutr. 30:443. *Quoted in* Underwood, E.J. 1971, p. 327.

Rosenfeld, I. and O.A. Beath. 1964. Selenium. Academic Press, New York, p. 411. *Quoted in* Moxon, Alvin L. and Oscar E. Olson 1974, p. 679.

Rusiecki, W. and J. Brzezinski. 1966. Influence of sodium selenate on acute thallium poisonings. Acta Pol. Pharm. 23:74. *Quoted in* Diplock, Anthony T. 1976.

Sakurai, Haruhiko and Kenzaburo Tsuchiya. 1975. A tentative recommendation for the maximum daily intake of selenium. Environ. Physiol. Biochem. 5:107-118.

Sandholm, M., H.E. Oksanen and L. Pesonen. 1973. Uptake of selenium by aquatic organisms. Limnol. Oceanogr. 18(3):496-499.

Schrauzer, G.N., D.A. White and C.J. Schneider. 1977a. Cancer mortality correlation studies–III: Statistical associations with dietary selenium intakes. Bioinorg. Chem. 7:23-34.

Schrauzer, G.N., D.A. White and C.J. Schneider. 1977b. Cancer mortality correlation studies–IV: Association with dietary intakes and blood levels of certain trace elements, notably Se-antagonists. Bioinorg. Chem. 7(1):35-56.

Schroeder, H.A. 1974. The Poisons Around Us. Toxic Metals in Food, Air, and Water. Indiana University Press, Bloomington.

Schutz, D.F. and K.K. Turekian. 1965. The investigation of the geographical and vertical distribution of several trace elements in sea water using neutron activation analysis. Geochim. Cosmochim. Acta, 29-259. *Quoted in* Lakin, Hubert W. 1973.

Schwarz, Klaus and Keshav D. Pathak. 1975. The biological essentiality of selenium and the development of biologically active organoselenium compounds of minimum toxicity. Chemica Scripta, 8A:85-95.

Scott, M.L. 1971. Role of selenium as an essential nutrient. *In* Newer Trace Elements in Nutrition. Edited by Walter Mertz and W.E. Cornatzer. Marcel Dekker, Inc., New York, pp. 51-56.

Sell, Jerry L. and Farouk G. Horani. 1976. Influence of selenium on toxicity and metabolism of methylmercury in chicks and quail. Fed. Proc. 35(3):577.

Shamberger, Raymond J. 1974. Antioxidants and cancer. III. Selenium and other antioxidants decrease carcinogen-induced chromosome breakage. *In* Trace Element Metabolism in Animals–2. Edited by W.G. Hoekstra, J.W. Suttie, H.E. Ganther, and W. Mertz. University Park Press, Baltimore. pp. 593-597.

Shamberger, Raymond J., Stanley Tytko and Charles Willis. 1973. Antioxidents and cancer. Selenium distribution and human cancer mortality in the United States, Canada and New Zealand. *In* Trace Substances in Environmental Health–VII. Proceedings of the 7th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia. Edited by Delbert D. Hemphill. pp. 35-40.

Shamberger, Raymond J., Stanley A. Tytko and Charles E. Willis. 1975. Selenium and heart disease. *In* Trace Substances in Environmental Health–IX. Proceedings of the 9th Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia. Edited by Delbert D. Hemphill. pp. 15-22.

Shamberger, Raymond J., Charles E. Willis and Lawrence J. McCormack. 1979. Blood selenium and heart mortality in 19 states. Part III, Selenium and heart disease. Paper presented at the 13th Annual Conference on Trace Metals in Environmental Health, Univ. of Missouri, Columbia.

Sielicki, Margarete and Jeffrey C. Burnham. 1973. The effect of selenite on the physiological and morphological properties of the blue-green alga *Phormidium luridum* var. *olivacea*. J. Phycol. 9(4):509-514.

Sillén, Lars Gunnar. 1963. How has seawater got its present composition? Sven. Kem. Tidskr. 75(4):161-177.

Spallholz, Julian E., Rollin H. Heinzerling, Marlene L. Gerlach and John L. Martin. 1973. Enhancement of immunological responses in mice by selenite. Fed. Proc. 32(3, Part 1):886.

Spallholz, Julian E., John L. Martin, Marlene L. Gerlach and Rollin H. Heinzerling. 1974. Effect of selenicals on the primary immune response in mice. *In* Trace Element Metabolism in Animals–2. Edited by W.G. Hoekstra, J.W. Suttie, H.E. Ganther, and W. Mertz. University Park Press, Baltimore, pp. 567-569.

Stadtman, Thressa C. 1966. Selenium biochemistry. Science, 183: 915-922.

Stijve, T. and R. Besson. 1976. Mercury, cadmium, lead and selenium content of mushroom species belonging to the genus *Agaricus*. Chemosphere, 5(2):151-158.

Taylor, S.R. 1964. Abundance of chemical elements in the continental crust: a new table. Geochim. Cosmochim, Acta, 28: 1273-1285.

Traversy, W.J., P.D. Goulden, Y.M. Sheikh and J.R. Leacock. 1975. Levels of arsenic and selenium in the Great Lakes Region. Environment Canada, Inland Waters Directorate, Scientific Series No. 58, Burlington, Ontario.

Turekian, K.K. and K.H. Wedepohl. 1961. Distribution of the elements in some major units of the earth's crust. Geol. Soc. Am. Bull. 72:175-192.

Underwood, E.J. 1971. Trace Elements in Human and Animal Nutrition. Academic Press, New York and London.

Underwood, E.J. 1976. Mineral imbalances in farm animals and their study and diagnosis with isotopic tracers. Atomic Energy Rev. 14(4):591-619.

Watkinson, J.H. 1974. New Zealand Med. J. 80:202. *Quoted in* Schrauzer, G.N., D.A. White, and C.J. Schneider 1977a.

Weir, Patricia A. and Charles H. Hine. 1970. Effects of various metals on behaviour of conditioned goldfish. Arch. Environ. Health, 29(1):45-51.

Weissman, G.S. and S.F. Trelease. 1955. Influence of sulfur on the toxicity of selenium to *Aspergillus*. Am. J. Bot. 42:489-495. *Quoted in* Niimi, A.J. and Q.N. LaHam 1976.

Weswig, Paul H. 1973. Selenium in the environment. *In* Heavy Metals in the Environment. Water Resources Research Institute. Oregon State University. pp. 183-203.

Wilson, Christine S. and Nicholas L. Petrakis. 1976. Selenium as a protective against breast cancer in non-western diets. Fed. Proc. 35(3):578.

Wood, G.E. 1975. Selenium and tellurium. *In* Canadian Minerals Yearbook, 1974. Department of Energy, Mines and Resources.

Wright, P.L. and M.C. Bell. 1966. Am. J. Physiol. 211:6. *Quoted in* Underwood, E.J. 1971, p. 330.

Zoller, W.H., E.S. Gladney and R.A. Duce. 1974. Atmospheric concentrations and sources of trace metals at the South Pole. Science, 183(4121):198-200.